```
     FILE 'CAPLUS, WPIDS, MEDLINE, EMBASE' ENTERED AT 11:06:32 ON 13 SEP 2003
L1        203773 S SELEN?
L2        248012 S SYSTEMIC INFLAMMATORY RESPONSE SYNDROME# OR SIRS OR ORGAN FAI
L3           124 S L1 (L) L2
L4            80 DUP REM L3 (44 DUPLICATES REMOVED)
L5            23 S PY>2001 AND L4
L6            57 S L4 NOT L5

=> d que
L1        203773 SEA SELEN?
L2        248012 SEA SYSTEMIC INFLAMMATORY RESPONSE SYNDROME# OR SIRS OR ORGAN
                 FAILURE# OR ORGAN DYSFUNCTION# OR MOF OR SOFA OR SEPSIS OR
                 SEPTIC SHOCK OR SEPTICEM? OR PERITONITIS OR PNEUMOPATH? OR
                 MENINGITIS
L3           124 SEA L1 (L) L2
L4            80 DUP REM L3 (44 DUPLICATES REMOVED)
L5            23 SEA PY>2001 AND L4
L6            57 SEA L4 NOT L5
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All Reviewed online

09/763,870

AN 1998182528 MEDLINE
DN 98182528 PubMed ID: 9522061
TI Effect of **selenium** supplementation on mice **infected** with LP-BM5 MuLV, a murine AIDS model.
AU Chen C; Zhou J; Xu H; Jiang Y; Zhu G
CS Department of Chemistry, Huazhong University of Science and Technology, Wuhan, PROC.
SO BIOLOGICAL TRACE ELEMENT RESEARCH, (1997 Winter) 59 (1-3) 187-93. Journal code: 7911509. ISSN: 0163-4984.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; AIDS
EM 199805
ED Entered STN: 19980520
Last Updated on STN: 19980520
Entered Medline: 19980508
AB LP-BM5 Murine leukemia **virus** (MuLV) **infection** of C57BL/6 mice develop a disease that has many features in common with human acquired immunodeficiency syndrome (AIDS), in particular abnormal lymphoproliferation and severe immunodeficiency. Thus, this MAIDS model may be useful for evaluation of potent antirival agents in vivo. Deficiency in antioxidant micronutrients such as **selenium**, zinc, and glutathione have been observed in AIDs and AIDS-related complex (ARC) patients. In the present study, the MAIDS model was used to evaluate immunological and oxidative effect of Se as sodium **selenite**. Results indicated that Se treatment 0.1 mg/kg/d (p.o.) inhibited splenomegaly and sera IgG elevation effectively. In addition to abnormal immunity, oxidative imbalance possibly existed in MAIDS model, as lipid peroxide increased significantly in spleen and whole blood glutathione peroxidase (GSH-Px) activity decreased markedly. Se supplementation had good protective effect.

0.1 mg/kg/day · viral infection

AN 1995:129229 CAPLUS
DN 122:104635
TI Antioxidant status of dairy cows **supplemented** prepartum with vitamin E and **selenium**
AU Brzezinska-Slebodzinska, E.; Miller, J. K.; Quigley, J. D., III; Moore, J. R.; Madsen, F. C.
CS Anim. Sci. Dep., Univ. Tennessee, Knoxville, TN, 37901-1071, USA
SO Journal of Dairy Science (1994), 77(10), 3087-95
CODEN: JDSCAE; ISSN: 0022-0302
DT Journal
LA English
AB Possible relationships among dietary antioxidants, oxidative status, and placental retention were investigated in periparturient dairy cows. During 6 wk prepartum, 16 cows each were given daily by capsule 1000 IU of vitamin E, 3 **mg** of **Se**, both vitamin E and Se, or neither (control). .alpha.-Tocopherol in serum and fast-acting antioxidants in plasma increased, but, in red blood cells, thiobarbituric acid-reactive substances decreased during the last 6 wk before parturition in cows given vitamin E. These measurements were unaffected by supplementation of Se. Cows that had retained placenta .gtoreq.12 h had lower fast-acting antioxidants in plasma and glutathione peroxidase in red blood cells up to 2 wk before calving than did cows that shed fetal membranes in <12 h. Results suggest that inadequate dietary antioxidants may increase oxidative stress, prodn. of lipid peroxides, and incidence of retained fetal membranes in dairy cows.

AN 1998:409527 CAPLUS
DN 129:131224
TI Sodium **selenite** and N-acetylcysteine in antiretroviral-naive HIV-I-**infected** patients: a randomized, controlled pilot study
AU Look, M. P.; Rockstroh, J. K.; Rao, G. S.; Barton, S.; Lemoch, H.; Kaiser, R.; Kupfer, B.; Sudhop, T.; Spengler, U.; Sauerbruch, T.
CS Departments of General Internal Medicine, University of Bonn, Bonn, 53105, Germany
SO European Journal of Clinical Investigation (1998), 28(5), 389-397
CODEN: EJCIB8; ISSN: 0014-2972
PB Blackwell Science Ltd.
DT Journal
LA English
AB The aim of this work was to study the effects of combined oral administration of N-acetylcysteine (NAC) and sodium **selenite** (Se) on plasma glutathione (GSH), lymphocyte subpopulations and viral load in asymptomatic human immunodeficiency **virus** (HIV)-**infected** patients. We used a prospective, randomized and controlled therapy trial with partial crossover. Twenty-four antiretroviral-naive HIV-**infected** outpatients at Centers for Disease Control (CDC) '93 stages I and II were randomized to receive the antioxidant combination NAC 600 mg t.i.d. and Se 500 .mu.g per day for either 24 wk (group A, n = 13) or from the end of week 12 (group B, n = 11) until the end of week 24. Thus, group B served as untreated control during the first 12 wk. There was (a) a trend towards an increase in the percentage of CD4+ lymphocytes after 6 wk (P = 0.08); (b) an increase in the CD4/CD8 ratio after 6 and 12 wk (P = 0.02 and P = 0.04 resp.); and (c) a decrease in the abs. CD8/CD38 count and percentage of lymphocytes after 6 wk (P = 0.002 and P = 0.033 resp.) and 12 wk (P = 0.033, P = 0.1 resp.) in group A compared with the control period of group B. The effects obsd. in group A were, however, not paralleled to the same extent by group B after crossing-over to treatment after 12 wk. In addn., erythrocyte glutathione peroxidase (GSH-Px) activity and GSH, glutathionedisulfide (GSSG) concns. and the reduced/total GSH ratio were not affected by the treatment. Serum **selenium** levels increased significantly (P < 0.001) upon treatment. Viral load was not altered. The changes in lymphocyte subsets after NAC/Se treatment were not comparable to those after std. antiretroviral drug therapy. This, however, does not preclude per se possible benefits of antioxidant supplementation in HIV disease.

Se — 500 µg/day → not enough

AN 1997:319803 CAPLUS
DN 127:49765
TI Protective role of selenium against hepatitis B virus and primary liver cancer in Qidong
AU Yu, Shu Yu; Zhu, Ya Jun; Li, Wen Gang
CS Cancer Institute, Chinese Academy of Medical Sciences, Peking Union Medical College, Beijing, 100021, Peop. Rep. China
SO Biological Trace Element Research (1997), 56(1), 117-124
CODEN: BTERDG; ISSN: 0163-4984
PB Humana
DT Journal
LA English
AB High rates of hepatitis B **virus** (HBV) **infection** and primary liver cancer (PLC) are present in Qidong county. Epidemiol. surveys demonstrated an inverse assocn. between **selenium** (Se) level and regional cancer incidence, as well as HBV **infection**. Four-year animal studies showed that dietary supplement of Se reduced the HBV **infection** by 77.2% and liver precancerous lesion by 75.8% of ducks, caused by exposure to natural environmental etiol. factors. An intervention trial was undertaken among the general population of 130,471. Individuals in five townships were involved for observation of the preventive effect of Se. The 8-yr follow-up data showed reduced PLC incidence by 35.1% in **selenized** table salt supplemented vs the nonsupplemented population. On withdrawal of Se from the treated group, PLC incidence rate began to increase. However, the inhibitory response to HBV was sustained during the 3-yr cessation of treatment. The clin. study among 226 Hepatitis B Surface Antigen (HBsAg)-pos. persons provided either 200 .mu.g of Se in the form of **selenized** yeast tablet or an identical placebo of yeast tablet daily for 4 yr showed that 7 of 113 subjects were diagnosed as having PLC in the placebo group, whereas no incidence of PLC was found in 113 subjects supplemented with Se. Again on cessation of treatment, PLC developed at a rate comparable to that in the control group, demonstrating that a continuous intake of Se is essential to sustain the chemopreventive effect.

Too Low

E.g. → 200 µg Se

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L1          28 SEA (SELENITE# OR SELENATE# OR SELENO? OR SELENIUM) (L)
               SYSTEMIC INFLAM? RESPONSE
L2          16 DUP REM L1 (12 DUPLICATES REMOVED)


L1          28 SEA (SELENITE# OR SELENATE# OR SELENO? OR SELENIUM) (L)
               SYSTEMIC INFLAM? RESPONSE
L2          16 DUP REM L1 (12 DUPLICATES REMOVED)
L3          31 SEA (SELENITE# OR SELENATE# OR SELENO? OR SELENIUM) (L)
               (PERITONITIS OR PNEUMOPATHY OR MENINGITIS OR SEPTICEMIA OR
               SEPTIC SHOCK)
L4          28 SEA L3 NOT L2
L5          17 DUP REM L4 (11 DUPLICATES REMOVED)


L1          28 SEA (SELENITE# OR SELENATE# OR SELENO? OR SELENIUM) (L)
               SYSTEMIC INFLAM? RESPONSE
L3          31 SEA (SELENITE# OR SELENATE# OR SELENO? OR SELENIUM) (L)
               (PERITONITIS OR PNEUMOPATHY OR MENINGITIS OR SEPTICEMIA OR
               SEPTIC SHOCK)
L6         327 SEA (SELENITE# OR SELENATE# OR SELENO? OR SELENIUM) (L)
               ((INFECT? (4A) (BACTERIA? OR PARASIT? OR FUNG? OR VIRUS? OR
               VIRAL)) OR RHEUMATOID POLYARTHRITIS)
L7         210 DUP REM L6 (117 DUPLICATES REMOVED)
L8         204 SEA L7 NOT (L1 OR L3)
L9         161 SEA L8 AND (SELEN?/AB OR INFECT?/AB OR POLYARTHRITIS/AB)
L10        112 SEA L9 AND (SELEN? (30A) (INFECT? OR POLYARTHRITIS))
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Reviewed online
Printed only few relevant hits

7/02

Files Caplus WPIDS medline Embase

for all queries

=> d 12 1-16 bib ab

L2 ANSWER 1 OF 16 WPIDS (C) 2002 THOMSON DERWENT
AN 2001-663083 [76] WPIDS
DNC C2001-194838
TI Preparation of enteral food material at the bed of critically ill patient, by providing standard enteral formulation, and adding to standard enteral formulation via closed system a composition(s) in module form.
DC B04 B05 D13
IN BALLEVRE, O; BOZA, J; BREUILLE, D; FINOT, P; JAUSSAN, V; ROESSLE, C; SCHWEIZER, T
PA (NEST) SOC PROD NESTLE SA
CYC 95
PI WO 2001078533 A2 20011025 (200176)* EN 20p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001056250 A 20011030 (200219)
ADT WO 2001078533 A2 WO 2001-EP3790 20010403; AU 2001056250 A AU 2001-56250 20010403
FDT AU 2001056250 A Based on WO 200178533
PRAI EP 2000-108412 20000418
AB WO 200178533 A UPAB: 20011227
NOVELTY - An enteral food material is prepared at the bed of a critically ill patient by i) providing a standard enteral formulation; and ii) adding to the standard enteral formulation via a closed system a composition(s) in a module form. The compositions contain nutrients, and are adapted for a specific clinical condition.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a nutritional module, optionally supplemented with carriers and/or excipients, for addition to standard enteral formula at the bed of a patient, consisting of the composition.
USE - The invention is used for preparing an enteral food material at the bed of a critically ill patient. The patient may be an individual suffering from multiple trauma, head injury, burns, sepsis, SIRS, or ARDS, or an individual who has been subjected to surgery (claimed).
ADVANTAGE - The invention addresses the changing nutritional needs of a patient and simultaneously avoids contamination of the enteral formulation by microorganisms.
Dwg.0/0

L2 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 1
AN 2001:756475 CAPLUS
DN 135:357212
TI The effect of a selenium supplementation on the outcome of patients with severe systemic inflammation, burn and trauma
AU Gartner, Roland; Albrich, Werner; Angstwurm, Matthias W. A.
CS Klinikum der Ludwig-Maximilians-Universitat Munchen, Medizinische Klinik-Innenstadt, Munchen, 80336, Germany
SO BioFactors (2001), 14(1-4), 199-204
CODEN: BIFAEU; ISSN: 0951-6433
PB IOS Press
DT Journal
LA English
AB Patients with **systemic inflammatory response** syndrome (SIRS) and sepsis exhibit decreased plasma **selenium** and glutathione peroxidase activity. This was shown in several clin. studies. Moreover, the degree of **selenium** deficiency correlates with the severity of the disease and the incidence of mortality. Patients with SIRS and sepsis are exposed to severe oxidative stress.

Selenoenzymes play a major role in protecting cells against peroxidn., esp. lipid peroxidn. and are involved in the regulation of inflammatory processes. Therefore, **selenium** substitution in those patients might be effective in the prevention of multiorgan failure. The results of randomized clin. trials investigating **selenium** substitution in crit. ill patients with inflammation are reviewed. In 2 independently performed randomized, prospective clin. trials, including patients with **systemic inflammatory response** syndrome or sepsis, the supplementation of **selenium** revealed a significant redn. in multiorgan failure and, esp., a lower incidence of acute renal failure and respiratory distress syndrome. One of those trials also could demonstrate a significant redn. of mortality in the most severely ill patients. Two other studies, where **selenium** together with other trace elements or a mixt. of antioxidants were used in the treatment of patients with severe burn injuries or trauma showed a significant redn. in the secondary infection rate, including sepsis. Thus, **selenium** supplementation seems to improve the outcome of patients with SIRS, sepsis and severe injury, however, pivotal prospective clin. trials with sufficient statistical power are now necessary to finally prove the efficacy of a **selenium** supplementation in these diseases.

RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 2000:161142 CAPLUS
DN 132:175825
TI Use of **selenium** compounds for treating patients suffering from **systemic inflammatory response** syndrome (SIRS), and composition for implementing the treatment
IN Forceville, Xavier; Vitoux, Dominique
PA Fr.
SO PCT Int. Appl., 25 pp.
CODEN: PIXXD2
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000012101	A2	20000309	WO 1999-FR2066	19990830
	WO 2000012101	A3	20000615		
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	FR 2782642	A1	20000303	FR 1998-10889	19980831
	FR 2782642	B1	20011207		
	AU 9954270	A1	20000321	AU 1999-54270	19990830
	BR 9913339	A	20010515	BR 1999-13339	19990830
	EP 1107767	A2	20010620	EP 1999-940254	19990830
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI	FR 1998-10889	A	19980831		
	WO 1999-FR2066	W	19990830		

AB The invention concerns the use of .gtoreq.1 **selenium**-contg. mols., in an amt. corresponding to a daily dose of about 2 to 40 mg, even 80 mg of at. **selenium** equiv., on its own or combined with other synergistic mols. for controlling oxidative stress and excessive

inflammatory reaction: zinc, vitamin E, vitamin C, iron chelators, glutathione precursors, copper and/or copper transport chelators, for prepg. a medicine for treating severe **systemic inflammatory response** syndrome, in particular any acute infectious condition endangering the patient's life whether of bacterial, parasitic, fungal or viral origin, and any condition corresponding to a severe onset of inflammatory pathol. bringing about an exacerbation of cytokine secretion. The invention is applicable in human and veterinary medicine. Use of sodium **selenite** in clin. situations is described.

L2 ANSWER 4 OF 16 WPIDS (C) 2002 THOMSON DERWENT
AN 2000-226349 [20] WPIDS
DNC C2000-069245
TI Treatment of severe **systemic inflammatory response** syndrome using sodium **selenite** or other **selenium** compound.
DC B06
IN FORCEVILLE, X; VITOUX, D
PA (FORC-I) FORCEVILLE X
CYC 89
PI FR 2782642 A1 20000303 (200020)* 13p
WO 2000012101 A2 20000309 (200020) FR
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW
AU 9954270 A 20000321 (200031)
BR 9913339 A 20010515 (200130)
EP 1107767 A2 20010620 (200135) FR
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
ADT FR 2782642 A1 FR 1998-10889 19980831; WO 2000012101 A2 WO 1999-FR2066 19990830; AU 9954270 A AU 1999-54270 19990830; BR 9913339 A BR 1999-13339 19990830, WO 1999-FR2066 19990830; EP 1107767 A2 EP 1999-940254 19990830, WO 1999-FR2066 19990830
FDT AU 9954270 A Based on WO 200012101; BR 9913339 A Based on WO 200012101; EP 1107767 A2 Based on WO 200012101
PRAI FR 1998-10889 19980831
AB FR 2782642 A UPAB: 20000426
NOVELTY - A **selenium**-containing compound is used for treating severe **systemic inflammatory response** syndrome (SIRS) or any state caused by a severe acute increase in cytokin secretion.

ACTIVITY - Antibacterial; immunosuppressive; antiinflammatory. A patient was admitted for post-operative resuscitation went into a state of shock (lactic acidosis) and suffered acute respiratory distress syndrome. He was given sodium **selenite** (4 mg Se/day) continuously for 24 hours, then 1 mg Se/day for the next 10 days. The lactic acidosis rapidly regressed, and he was able to leave resuscitation after 10 days, resuming a normal life within 3 months.

MECHANISM OF ACTION - None given.

USE - Treatment of septic shock, peritonitis, pneumopathia, meningitis and bacterial septicemia.

Dwg.0/0

L2 ANSWER 5 OF 16 MEDLINE
AN 1999435481 MEDLINE
DN 99435481 PubMed ID: 10507647
TI **Selenium** replacement in severe **systemic inflammatory response** syndrome.

AU Opal S M
SO CRITICAL CARE MEDICINE, (1999 Sep) 27 (9) 2042-3.
Journal code: 0355501. ISSN: 0090-3493.
CY United States
DT Editorial
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199910
ED Entered STN: 19991101
Last Updated on STN: 19991101
Entered Medline: 19991015

L2 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1999:468053 CAPLUS
DN 131:111450
TI Mercapto and seleno derivatives as inhibitors of nitric oxide synthase
IN Southan, Garry J.; Salzman, Andrew L.; Szabo, Csaba
PA Children's Hospital Medical Center, USA
SO U.S., 16 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5929063	A	19990727	US 1995-545952	19951020
	US 5674907	A	19971007	US 1995-410312	19950324
	CA 2214601	AA	19961003	CA 1996-2214601	19960322
	WO 9630007	A1	19961003	WO 1996-US3838	19960322
	W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML				
	AU 9653191	A1	19961016	AU 1996-53191	19960322
	AU 695307	B2	19980813		
	EP 814792	A1	19980107	EP 1996-909808	19960322
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	CN 1181700	A	19980513	CN 1996-192791	19960322
	JP 11502847	T2	19990309	JP 1996-529506	19960322
	BR 9607951	A	19990601	BR 1996-7951	19960322
	US 5952385	A	19990914	US 1997-889379	19970708
	AU 9892381	A1	19990114	AU 1998-92381	19981116
	AU 729933	B2	20010215		
	US 5985917	A	19991116	US 1999-281125	19990329
PRAI	US 1995-410312	A2	19950324		
	US 1995-545952	A	19951020		
	AU 1996-53191	A3	19960322		
	WO 1996-US3838	W	19960322		

OS MARPAT 131:111450
AB A pharmacol. acceptable compn. is provided for inhibiting nitric oxide synthase in a mammal, which includes a mercapto or seleno deriv. and a pharmaceutically acceptable carrier. The invention also concerns a method of inhibiting nitric oxide synthase, selectively inhibiting the inducible isoform of nitric oxide synthase, and treating various conditions where there is an advantage in inhibiting nitric oxide biosynthesis. The method includes the step of administering to a mammal a mercapto or seleno deriv. in pure form or in a pharmaceutically acceptable carrier.
RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 7 OF 16 WPIDS (C) 2002 THOMSON DERWENT
AN 1999-305285 [26] WPIDS
DNC C1999-089780
TI Formulation for treatment of e.g. liver disorders includes selenium, vitamins A, C and E, amino acid and coenzyme Q10.
DC B05
IN HENRIKSEN, B
PA (PHAR-N) PHARMA NORD UK LTD; (PHAR-N) PHARMA NORD APS
CYC 27
PI GB 2330531 A 19990428 (199926)* 15p
EP 913155 A2 19990506 (199926) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
JP 11199477 A 19990727 (199940) 7p
US 6136859 A 20001024 (200055)
EP 913155 B1 20020320 (200221) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
DE 69804281 E 20020425 (200235)
GB 2330531 B 20020605 (200238)
ADT GB 2330531 A GB 1998-23038 19981022; EP 913155 A2 EP 1998-308654 19981022; JP 11199477 A JP 1998-304137 19981026; US 6136859 A US 1998-177555 19981023; EP 913155 B1 EP 1998-308654 19981022; DE 69804281 E DE 1998-604281 19981022, EP 1998-308654 19981022; GB 2330531 B GB 1998-23038 19981022
FDT DE 69804281 E Based on EP 913155
PRAI GB 1997-22361 19971024
AB GB 2330531 A UPAB: 20011211
NOVELTY - Formulation comprising organic or inorganic **selenium**, beta -carotene and/or vitamin A, ascorbic acid or its salt or ester, alpha -tocopherol or its derivative, methionine and coenzyme Q10 (ubiquinone) together with a carrier, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is provided for the use of organic or inorganic **selenium** in combination with beta -carotene, ascorbic acid or its salt or ester, alpha -tocopherol or its derivative, methionine and coenzyme Q10 for the treatment of biliary cirrhosis.

ACTIVITY - Hepatotropic; virucide; antiinflammatory; antiulcer; nootropic; anticonvulsant; cardiant; ophthalmological; antiparkinsonian; cerebroprotective; antiarthritic.

24 Patients (mean age 61.3 plus or minus 9.4 years) who were anti-mitochondrial antibody positive and at various stages of primary biliary cirrhosis, were assessed for pruritis and fatigue, and were then randomly assigned to receive either vitamin, trace elements and sulphur containing amino acids formulation (group A) or vitamin, trace elements, sulphur containing amino acids and coenzyme Q10 (group B). After 3 months, fatigue and pruritis were again assessed and significant symptomatic improvements were observed. Itch, assessed on a scale of 0 (no problem) to 4 (extreme problem) was rated as 3.3 plus or minus 4.2 before and 2.5 plus or minus 3.2 after treatment for group A and 2.4 plus or minus 3.0 before and 0.4 plus or minus 0.7 after treatment for group B. Night itch, assessed on a scale of 1 (sleep not disturbed) to 6 (sleep disturbed every night) was rated as 3.0 plus or minus 2.3 before and 1.9 plus or minus 1.6 after treatment for group A and 2.6 plus or minus 1.9 before and 1.3 plus or minus 0.7 after treatment for group B. Fatigue, assessed using the Fisk fatigue impact score (score out of 160, reduction indicating therapeutic benefit) was 43.7 plus or minus 32.5 before and 39.2 plus or minus 40.6 after treatment for group A and 60.3 plus or minus 49.3 before and 40.3 plus or minus 37.5 after treatment for group B.

MECHANISM OF ACTION - None given.

USE - The formulation is useful for the treatment of primary biliary cirrhosis (PBC), viral hepatitis, steatohepatitis, alcoholic cirrhosis and related hepatic and biliary disorders, **systemic inflammatory response** syndrome (SIRS) leading to

multiple organ dysfunction syndrome (MODS), inflammatory bowel diseases e.g. colitis, Crohn's disease and ulcerative colitis, mitochondrial diseases e.g. Huntington's chorea and Leigh's disease, fibromyalgia, pancreatitis, fatigue syndromes and disorders where an excess of free radicals may play a causative role e.g. myocardial infarction, cataract formation, Parkinson's disease, stroke or arthritis.

ADVANTAGE - None given.

L2 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2
AN 1999:790733 CAPLUS
DN 132:234923
TI Oxidative stress in acute pancreatitis
AU Schulz, Hans-Ulrich; Niederau, Claus; Klonowski-Stumpe, Hanne; Halangk, Walter; Luthen, Reinhardt; Lippert, Hans
CS Department of Surgery, Otto-von-Guericke-University of Magdeburg, Magdeburg, D - 39120, Germany
SO Hepato-Gastroenterology (1999), 46(29), 2736-2750
CODEN: HEGAD4; ISSN: 0172-6390
PB H.G.E. Update Medical Publishing
DT Journal; General Review
LA English
AB A review with 137 refs. The present work critically reviews the evidence for an involvement of free radicals in the pathophysiol. of acute pancreatitis and the potential of treatment with antioxidants and scavenger substances. Data originating from clin. trials, exptl. pancreatitis studies and in vitro investigations are included. Enhanced free radical activities and increased concns. of lipid peroxides in plasma and tissue have been found in both patients and exptl. animals with acute pancreatitis. The individual contribution of possible sources of free radicals (e.g., invading inflammatory cells, xanthine oxidase, cytochromes P 450, nitric oxide synthase) is not yet clear, however. Since prophylactic administration of antioxidants diminished, in particular, pancreatic edema formation, free radicals seem to play an important role in the genesis of edema in acute pancreatitis. An involvement of free radicals in the pathogenesis of pancreatic necrosis could not yet be proven. Thus, no antioxidant treatment has proven useful for therapy of fulminant pancreatitis in animals to date. However, in severe acute pancreatitis characterized by death occurring after 12-18 h, the **seleno**-org. compd. Ebselen, which has a glutathione peroxidase-like activity, and the membrane permeable ascorbic acid deriv. CV-3611 have been demonstrated to be effective. To date, controlled clin. studies have failed to demonstrate the therapeutic efficacy of antioxidant **selenium** or glutathione precursor supplementation. Therefore, further controlled clin. trials are needed to det. whether supplements of antioxidants can alter the clin. course of acute pancreatitis. Since the nitric oxide radical may even protect the pancreas, a purely neg. discussion of the role of free radicals on the pancreas is not justified. The actual role of free radicals in acute pancreatitis, i.e. serving the body's defense against infection, being an epiphenomenon of the inflammatory process without pathophysiol. relevance, or having true pathogenic significance, is not yet clear. Lipid peroxidn. may perhaps not be the cause but rather the sequel of pancreatic inflammation and may likely reflect the severity of the **systemic inflammatory response** rather than that of pancreatic parenchyma damage. In vitro, exposure of isolated pancreatic acinar cells to oxidative stress caused rapid cell damage and death. Such knowledge from cellular studies might help to plan therapeutical trials to evaluate potentially effective therapies in the exptl. animal, as well as in patients suffering from pancreatitis. Thus, to further clarify the role of oxidative stress in acute pancreatitis, an integrated approach is needed, including investigations at various biol. levels, from isolated cells or even organelles to lab. animals and, finally, clin. studies in man.
RE.CNT 137 THERE ARE 137 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 9 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 1999337903 EMBASE
TI **Selenium** replacement in severe **systemic inflammatory response** syndrome.
AU Opal S.M.
CS Dr. S.M. Opal, Memorial Hospital of Rhode Island, Infectious Disease Section, 111 Brewster Street, Pawtucket, RI 02860, United States
SO Critical Care Medicine, (1999) 27/9 (2042-2043).
Refs: 10
ISSN: 0090-3493 CODEN: CCMDC7
CY United States
DT Journal; Editorial
FS 005 General Pathology and Pathological Anatomy
006 Internal Medicine
029 Clinical Biochemistry
037 Drug Literature Index
LA English

L2 ANSWER 10 OF 16 MEDLINE DUPLICATE 3
AN 1999435436 MEDLINE
DN 99435436 PubMed ID: 10507602
TI **Selenium** replacement in patients with severe **systemic inflammatory response** syndrome improves clinical outcome.
AU Angstwurm M W; Schottdorf J; Schopohl J; Gaertner R
CS Intensive Care Unit, Klinikum Innenstadt, University of Munich, Department of Internal Medicine, Germany.
SO CRITICAL CARE MEDICINE, (1999 Sep) 27 (9) 1807-13.
Journal code: 0355501. ISSN: 0090-3493.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199910
ED Entered STN: 19991101
Last Updated on STN: 19991101
Entered Medline: 19991015
AB OBJECTIVE: To determine the effect of **selenium** replacement on morbidity and mortality in patients with **systemic inflammatory response** syndrome (SIRS). DESIGN: Controlled, randomized prospective open-label pilot study comparing patients with and without **selenium** replacement. SETTING: Intensive care unit of a university hospital for internal medicine. PATIENTS: Forty-two patients with SIRS caused by infection and a minimal Acute Physiology and Chronic Health Evaluation (APACHE) II score of 15 points on the day of admission were included. The **selenium** replacement group of patients (Se+; n = 21) received sodium **selenite** for 9 days (535 microg [6.77 micromol] for 3 days, 285 microg [3.61 micromol] for 3 days, and 155 microg [1.96 micromol] for 3 days) and thereafter, 35 microg (0.44 micromol) per day iv. The control group (Se-, n = 21) received 35 microg of sodium **selenite** throughout the total treatment period. INTERVENTIONS: Morbidity and clinical outcome was monitored by scoring using the APACHE III score, occurrence of acute renal failure, need and length of mechanical ventilation, and hospital mortality. Blood samples on days 0, 3, 7, and 14 were analyzed for serum **selenium** concentration and glutathione peroxidase (GSH-Px) activity. MEASUREMENTS AND MAIN RESULTS: The median APACHE II score at admission, age, gender, underlying diseases, serum **selenium** levels, and GSH-Px activities at admission were identical

in both groups. In Se+ patients, serum **selenium** levels and GSH-Px activity normalized within 3 days, whereas in controls, both variables remained significantly low ($p < .0001$). The APACHE III score decreased significantly in both groups but was significantly lower in the Se+ group (day 3, $p > .05$; day 7, $p = .018$; and day 14, $p = .045$ Se+ compared with Se-). Hemodialysis with continuous veno-venous hemodialysis because of acute renal failure was necessary in nine Se- compared with three Se+ patients ($p = .035$). Overall mortality in the Se- group was 52% vs. 33.5% in the Se+ group ($p = .13$). CONCLUSIONS: **Selenium** replacement in patients with SIRS seems to improve clinical outcome and to reduce the incidence of acute renal failure requiring hemodialysis.

L2 ANSWER 11 OF 16 MEDLINE DUPLICATE 4
AN 2000022192 MEDLINE
DN 20022192 PubMed ID: 10554541
TI [Selenium administration in children with SIRS].
Selensubstitution bei Kindern mit SIRS.
AU Borner J; Zimmermann T; Albrecht S; Roesner D
CS Klinik und Poliklinik fur Kinderchirurgie, Universitatsklinikum Carl Gustav Carus, TU Dresden.. Jens.Boerner@mailbox.tu-dresden.de
SO MEDIZINISCHE KLINIK, (1999 Oct 15) 94 Suppl 3 93-6.
Journal code: 8303501. ISSN: 0723-5003.
CY GERMANY: Germany, Federal Republic of
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals
EM 200001
ED Entered STN: 20000114
Last Updated on STN: 20000114
Entered Medline: 20000106
AB PATIENTS AND METHOD: At the Clinic for Paediatric Surgery of the University of Dresden, in a time period ranging from 5/1994 to 12/1996, all patients aged between 1 and 16 years with severe inflammatory surgical diseases or extended scalded skin, were given an adjuvant **selenium** substitution. As control group, all patients with the same diagnosis and age treated during the months 1/1997 to 12/1998, did not receive this adjuvant **selenium** substitution. All these patients fulfilled the criteria of **"Systemic Inflammatory Response** Syndrome" (SIRS). The **selenium**-therapy group consisted of 34 patients and the control group without substitution consisted of 31 patients. The following laboratory parameters were measured on the 1st, 2nd, 3rd, 6th and last treatment day: white blood cell count, interleukin 6, C-reactive protein, fibrinogen, malondialdehyde, activity of glutathione peroxidase in plasma and level of **selenium** in plasma and whole blood. RESULTS: The initially high interleukin 6 rates declined significantly in both groups from the 2nd day on. The acute phase proteins, i.e. the C-reactive protein and fibrinogen, normalized in both groups after the 3rd day of treatment. The initial low rates of **selenium** in plasma and blood gained more rapidly a normal level in the therapy group than in the control group. On the 1st day of therapy the glutathione peroxidase activity in plasma was in both groups at the inferior limit of norm range and remained at this level in the control group for the whole observation period. In the **selenium** -substitution group on the contrary, these initial low values raised to the double as an expression of an elevated cell membrane protection. The initial significant elevated malondialdehyde rates in both groups, expressing a raised lipidperoxidation, fell down to a normal level in the **selenium**-substitution group, whereas they remained at their initial high level in the control group during the whole observation period. CONCLUSION: The substitution of **selenium** in children with SIRS is a supportive therapy.

L2 ANSWER 12 OF 16 MEDLINE DUPLICATE 5
AN 2000022182 MEDLINE
DN 20022182 PubMed ID: 10554531
TI [Significance of selenium in intensive care medicine. Clinical studies of patients with SIRS/sepsis syndrome].
Die Bedeutung von Selen in der Intensivmedizin. Klinische Studien bei Patienten mit SIRS/Sepsis.
AU Gartner R; Angstwurm M
CS Medizinische Klinik, Klinikum Innenstadt, Ludwig-Maximilians-Universitat Munchen.. rgartner@medinn.med.uni-muenchen.de
SO MEDIZINISCHE KLINIK, (1999 Oct 15) 94 Suppl 3 54-7. Ref: 39
Journal code: 8303501. ISSN: 0723-5003.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA German
FS Priority Journals
EM 200001
ED Entered STN: 20000114
Last Updated on STN: 20000114
Entered Medline: 20000106
AB **Selenium** is an essential component of the intracellular antioxidant system as a structural component of the active center of the glutathione peroxidase enzymes. These **selenoenzymes** play a major role in protecting cells against peroxidation, especially lipid peroxidation and **selenium** seems to play a direct role in the regulation of inflammatory processes. In conditions of **systemic inflammatory response** or sepsis, patients are exposed to severe oxidative stress. These patients already have both, a decreased plasma **selenium** and glutathione peroxidase activity at admission to the ICU as has been shown in several studies. The degree of **selenium** deficiency is correlated with the severity of disease and the incidence of mortality. The reason for the low plasma **selenium** levels is unknown. Especially it would be of interest a) if the low plasma **selenium** is the consequence of the **systemic inflammatory response** with distribution of **selenium** in other compartments of the body, b) most important, whether the substitution of **selenium** might improve the outcome and decrease the mortality rate of these patients. In 2 independently performed intention-to-treat studies including patients with **systemic inflammatory response** syndrome or sepsis a beneficial effect of **selenium** supplementation on multiple organ function and outcome could already be demonstrated as well as a tendency of an improved mortality rate. A prospective analytical study clearly could demonstrate the inverse relationship between low plasma **selenium** and morbidity and mortality of patients with SIRS/sepsis. The results of these studies are so convincing, that we propose a randomized, prospective, double blind multicenter phase-III study including patients with **systemic inflammatory response** syndrome or sepsis to investigate, whether a high-dose **selenium** substitution in addition to the recommended treatment strategies for patients with sepsis improves outcome and mortality rate of these patients.

L2 ANSWER 13 OF 16 MEDLINE DUPLICATE 6
AN 1998422210 MEDLINE
DN 98422210 PubMed ID: 9751590
TI Selenium, systemic immune response syndrome, sepsis, and outcome in critically ill patients.
CM Comment in: Crit Care Med. 1998 Sep;26(9):1478-9
AU Forceville X; Vitoux D; Gauzit R; Combes A; Lahilaire P; Chappuis P
CS Department of Medical and Surgical Intensive Care, Centre Hospitalier de

Meaux, France.
SO CRITICAL CARE MEDICINE, (1998 Sep) 26 (9) 1536-44.
Journal code: 0355501. ISSN: 0090-3493.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199810
ED Entered STN: 19981020
Last Updated on STN: 19981020
Entered Medline: 19981008
AB OBJECTIVES: To confirm early, marked decrease in plasma **selenium** concentrations in patients admitted to a surgical and medical intensive care unit (ICU), and to study this decrease according to the presence or absence of **systemic inflammatory response** syndrome (SIRS), sepsis, or direct ischemia-reperfusion. DESIGN: Prospective, observational study. SETTINGS: Collaboration between the adult ICU of a 1,100-bed general hospital and a biochemical research laboratory of a university medical center. PATIENTS: One hundred thirty-four consecutive surgical and medical ICU patients. INTERVENTIONS: None. MEASUREMENTS AND MAIN RESULTS: In the first 31 patients, plasma and urine **selenium** concentrations were measured by electrothermal atomic absorption spectrometry on admission and once weekly during their ICU stay. These values were compared first with severity scores, criteria for SIRS, sepsis, and organ system failure taken on admission, and then with nosocomial infection, organ system failure during ICU stay, and hospital mortality. An early, low mean plasma **selenium** concentration was observed in these patients compared with **selenium** laboratory reference values. Plasma **selenium**, measured on ICU admission, inversely correlated with Acute Physiology and Chronic Health Evaluation II or Simplified Acute Physiology II scores. Patients with SIRS had lower **selenium** concentrations than those without SIRS. Mean urine **selenium** losses were normal in the first 31 patients. Plasma **selenium** concentration was low in all patients with severe sepsis and septic shock (range 0.20 to 0.72 micromol/L) and in those patients with ischemia-reperfusion from aortic cross-clamping (range 0.34 to 0.68 micromol/L). Despite recommended specific **selenium** supplementation, plasma **selenium** concentrations remained low for >2 wks in patients with SIRS. However, there was a slight increase in plasma **selenium** concentrations in surviving SIRS patients, whereas plasma **selenium** concentrations decreased in nonsurviving patients. The frequency of ventilator-associated pneumonia, organ system failure, and mortality was three times higher in patients with low plasma **selenium** concentration at the time of admission (**selenium** < or =0.70 micromol/L) than for the other patients. CONCLUSIONS: In severely ill ICU patients with SIRS, we observed an early 40% decrease in plasma **selenium** concentrations, reaching values observed in deleterious nutritional **selenium** deficiency. This prolonged decrease in **selenium** concentrations could explain the three-fold increase in morbidity and mortality rates in these patients compared with other ICU patients. The efficacy of **selenium** treatment in SIRS patients with a high gravity index score or hypoperfusion needs further investigation.

L2 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 7
AN 1997:664594 CAPLUS
DN 127:287894
TI Substitution of selenium in patients with severe inflammatory disease or with burns in childhood
AU Borner, Jens; Zimmermann, Thomas; Albrecht, Steffen; Roesner, Dietmar
CS Klinik Poliklinik Kinderchirurgie, Klinikum Carl Gustav Carus, Dresden, D-01307, Germany
SO Medizinische Klinik (Munich) (1997), 92(Suppl. 3), 17-19

CODEN: MEKLA7; ISSN: 0723-5003
PB Urban & Vogel
DT Journal
LA German
AB Effects of Se substitution were investigated in young patients with systematic inflammatory response syndrome (SIRS) or with burns on white cell count, interleukin 6, C-reactive protein, fibrinogen, malondialdehyde, activity of glutathione peroxidase in plasma, and Se levels in plasma and whole blood. Patients with low Se levels reached normal Se values more quickly with Se substitution. Elevated values of malondialdehyde as sign of increased peroxidn. of lipids normalized by Se substitution. Low activity of Se level in plasma was increased under Se substitution as sign of increased protection of the cell membrane.

L2 ANSWER 15 OF 16 MEDLINE DUPLICATE 8
AN 1998002387 MEDLINE
DN 98002387 PubMed ID: 9417494
TI [Selenium administration in patients with sepsis syndrome. A prospective randomized study].
Selensubstitution bei Sepsispatienten. Eine prospektiv randomisierte Studie.
AU Zimmermann T; Albrecht S; Kuhne H; Vogelsang U; Grutzmann R; Kopprasch S
CS Klinik fur Viszeral-, Thorax- und Gefasschirurgie, Universitatsklinikums Carl Gustav Carus der TU Dresden.
SO MEDIZINISCHE KLINIK, (1997 Sep 15) 92 Suppl 3 3-4.
Journal code: 8303501. ISSN: 0723-5003.
CY GERMANY: Germany, Federal Republic of
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA German
FS Priority Journals
EM 199712
ED Entered STN: 19980116
Last Updated on STN: 19980116
Entered Medline: 19971224
AB PATIENTS AND METHOD: In this study the effect of antioxidative therapy with sodium **selenite** was investigated in patients with **systemic inflammatory response** syndrome (S. I. R. S.) and multiple organ failure. 40 patients were included in this prospective randomized study. The patients were observed over a period of 28 days. The letality rate within 28 days was excepted as main criteria. The Apache-II and the MOF-Score of Goris were used as clinical parameters. 20 patients were treated with sodium **selenite** over a period of 28 days. RESULT: This antioxidative therapy reduced the letality rate from 40 to 15%.

L2 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 9
AN 1996:721653 CAPLUS
DN 126:1215
TI Mercapto and seleno derivatives as inhibitors of nitric oxide synthase
IN Southan, Garry J.; Salzman, Andrew L.; Szabo, Csaba
PA Children's Hospital Medical Center, USA
SO PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9630007	A1	19961003	WO 1996-US3838	19960322

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT,

LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML

US	5674907	A	19971007	US 1995-410312	19950324
US	5929063	A	19990727	US 1995-545952	19951020
AU	9653191	A1	19961016	AU 1996-53191	19960322
AU	695307	B2	19980813		
EP	814792	A1	19980107	EP 1996-909808	19960322
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP	11502847	T2	19990309	JP 1996-529506	19960322
BR	9607951	A	19990601	BR 1996-7951	19960322
PRAI US	1995-410312	A	19950324		
US	1995-545952	A	19951020		
WO	1996-US3838	W	19960322		

OS MARPAT 126:1215

AB Pharmacol. acceptable compns. are disclosed for inhibiting nitric oxide synthase in a mammal; the compns. include a mercapto or seleno deriv. and a pharmaceutically acceptable carrier. Also disclosed is a method of inhibiting nitric oxide synthase, selectively inhibiting the inducible isoform of nitric oxide synthase, and treating various conditions where there is an advantage in inhibiting nitric oxide biosynthesis. The method includes the step of administering to a mammal a mercapto or seleno deriv. in pure form or in a pharmaceutically acceptable carrier.

L5 ANSWER 1 OF 17 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 1
AN 2002:366358 CAPLUS
TI Serum and ascitic fluid selenium levels in patients with cirrhosis
AU Sancak, B.; Ozenirler, S.; Coskun, U.; Candan, S.; Unal, A.; Maral, I.
CS Department of Biochemistry, Faculty of Medicine, Gazi University, Ankara, TR 06510, Turk.
SO Trace Elements and Electrolytes (2002), 19(2), 82-86
CODEN: TEELEO; ISSN: 0946-2104
PB Dustri-Verlag Dr. Karl Feistle
DT Journal
LA English
AB The aim of this study was to det. **selenium** levels in cirrhotic patients and to investigate whether the existence of spontaneous bacterial **peritonitis** (SBP) and the degree of liver cirrhosis had an effect on serum and ascitic fluid **selenium** (Se) levels in cirrhotic patients. Serum and ascitic fluid **selenium** levels were studied in 32 cirrhotic patients and 10 healthy controls. Patients were divided into 4 groups. Control subjects (group I, n = 10), patients with compensated cirrhosis (group II, n = 16), patients with massive ascites (group III, n = 14), patients with massive ascites and spontaneous bacterial **peritonitis** (SBP) (group IV, n = 13). Serum **selenium** was analyzed by at. absorption spectrophotometry using an Unicam 939 AA Spectrometer, equipped with Unicam VP 90 vapor system. All cirrhotic patients (groups II, III, IV) showed significant decrease in serum **selenium** levels in comparison with that in control subject (group I) ($p < 0.05$). Although serum **selenium** levels were higher (group II: 46 .+-. 16.0 ng/mL) in patients with compensated cirrhosis when compared with other cirrhotic patients (group III: 42.9 .+-. 11.0 ng/mL, group IV: 38.4 .+-. 6.6 ng/mL), they were not statistically significant ($p > 0.05$). Ascitic fluid **selenium** levels were not different between decompensated cirrhotic patients with or without SBP (group III: 10.9 .+-. 5.4 ng/mL, group IV: 14.9 .+-. 7.3 ng/mL) ($p > 0.05$). Our findings suggest that decreased serum **selenium** levels in cirrhotic patients are not related to the degree of liver cirrhosis and spontaneous bacterial **peritonitis**.
RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 17 MEDLINE DUPLICATE 2
AN 2002144151 MEDLINE
DN 21867855 PubMed ID: 11878087
TI [**Septic shock** and **selenium** administration].
Choc septique et administration de **selenium**.
AU Forceville X; Aouizerate P; Guizard M
CS Centre Hospitalier de Meaux, 6-8 rue Saint-Fiacre, BP 218, 77104 Meaux, France.
SO THERAPIE, (2001 Nov-Dec) 56 (6) 653-61. Ref: 56
Journal code: 0420544. ISSN: 0040-5957.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA French
FS Priority Journals
EM 200204
ED Entered STN: 20020307
Last Updated on STN: 20020425
Entered Medline: 20020424
AB Selenium is an essential trace element. In the form of selenocysteine, an amino acid, selenium is necessary for the activity of important enzymes (i.e. glutathione peroxidases, thioredoxin reductase). In the periodic table of the elements, selenium belongs to the same column as oxygen. In

fact, seleno-enzymes have an important role in the detoxification of reactive oxygen species, especially peroxides and hydroperoxides. In septic and septic-like shock patients, reactive oxygen species, particularly peroxides, play an important role through their destructive actions, which are favourable as critical components of microbial destruction and also deleterious in excessive generation. This excessive generation results in tissue damage. Moreover, reactive oxygen species modulate the activation of important intracellular mediators (NF kappa B activation, arachidonic acid cascade). Simultaneously in patients with severe infection, there is a marked and early plasma selenium decrease. Redistribution due to selective selenium uptake for metabolic use could be one of the main mechanisms for this decrease. This review was carried out by questioning on the one hand the Medline database, by consulting the reviews and works available in the services of biology, biochemistry and pharmacy, by a prospective follow-up on the subject in Current Contents, but also thanks to library searches carried out by Aguettant laboratories. Several supplementary studies at various doses (from 140 to 1000 micrograms/day sodium selenite) have been conducted, though only on small groups of patients and with a questionable design. Selenium treatment seem to be promising in severely septic patients. However, in the absence of pertinent clinical data, only the administration of doses below adverse effect levels, staying within physiological limits, can presently be recommended (i.e. 200 to 500 micrograms/day of sodium selenite).

L5 ANSWER 3 OF 17 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3
AN 2000:717334 CAPLUS
DN 134:85418
TI Effect of selenium deficiency on the development of central nervous system lesions in murine listeriosis
AU Altimira, J.; Prats, N.; Lopez, S.; Domingo, M.; Briones, V.; Dominguez, L.; Marco, A.
CS Departamento de Patologia y Producciones Animales (Histologia y Anatomia Patologica), Facultad de Veterinaria, Universidad Autonoma de Barcelona, Barcelona, 08193, Spain
SO Journal of Comparative Pathology (2000), 123(2-3), 104-109
CODEN: JCVPAR; ISSN: 0021-9975
PB W. B. Saunders Co. Ltd.
DT Journal
LA English
AB The effect of **selenium** (Se) deficiency, produced by feeding a Se-deficient diet, on the development of central nervous system (CNS) lesions was studied in mice infected with Listeria monocytogenes, administered in drinking water for 1 or 7 days in a daily dose of 109 organisms, or for 7 days in a daily dose of 107. Se-deficient mice differed from Se-normal controls in developing CNS lesions significantly more frequently. Moreover, regardless of Se status, mice receiving repeated doses of 109 organisms differed from those receiving a single 109 dose in showing CNS lesions at least twice as often. The majority of animals with CNS lesions showed an inflammatory pattern of rhombencephalitis (17/24), while only two of 24 showed choroiditis-ventriculitis-**meningitis**; five of 24 animals showed both inflammatory patterns. Listeria monocytogenes antigen was identified within the areas of inflammation by an immunoperoxidase technique. Neuritis of the trigeminal nerve was present in eight animals. The relative lack of pathol. changes in the liver and spleen validates this murine model for the study of CNS listeriosis.
RE.CNT 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 17 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 1999348886 EMBASE
TI [Lipoperoxidation and antioxidatory protection of an organism during weaning from mechanical ventilation].

LIPOPEROXIDACE A ANTIOXIDATIVNI OCHRANA ORGANISMU V PRUBEHU ODVYKANI OD VENTILATORU.

AU Cerny V.; Zivny P.; Dostal P.; Parizkova R.

CS Dr. V. Cerny, E. Benese 1537, 500 12 Hradec Kralove, Czech Republic

SO Anesteziologie a Neodkladna Pece, (1999) 10/5 (203-209).
Refs: 15
ISSN: 0862-4968 CODEN: ANPEFF

CY Czech Republic

DT Journal; Article

FS 024 Anesthesiology

LA Czech

SL English; Czech

AB According to the current literature data, free oxygen radicals and mechanism of lipoperoxidation play an important role during development of muscular system dysfunction during sepsis and **septic shock**. Muscular dysfunction can affect respiratory muscles and contribute to muscular fatigue with subsequent need for ventilatory support. The aim of the study was to assess the degree of lipoperoxidation and capacity of antioxidatory apparatus in patients during weaning period. In 37 mechanically ventilated patients we prospectively monitored the concentrations of malonedialdehyd, glutathion, glutathionperoxidase activity and superoxiddismutase activity; betacaroten concentrations and **selenium** concentrations. The values were obtained on admission, last day of mechanical ventilation, at the start of weaning and after 24 hours of spontaneous breathing after disconnecting from ventilatory support. According to the length of weaning, patients were divided into two groups: group S, weaning period .ltoreq. 3 days, n = 15; group L, weaning period > 3 days, n = 22. Patients weaned for more than three days had significantly higher concentrations of malonedialdehyd on admission, significantly lower activity of glutathionperoxidase level when successfully weaned, non- significantly lower levels of beta-caroten and **selenium**. Prolonged ventilatory support and weaning period longer than three days were associated with higher degree of lipoperoxidation on admission and with a decrease of concentrations of selected markers of antioxidatory protective mechanisms. The results support an assumption that lipoperoxidation may play a role in the development of muscular system dysfunction in patients during the weaning period.

L5 ANSWER 5 OF 17 WPIDS (C) 2002 THOMSON DERWENT

AN 1999-095313 [08] WPIDS

CR 1999-095304 [08]; 2000-431259 [36]; 2001-181513 [09]

DNC C1999-028059

TI New isoquinoline-indole derivatives - used for treating bacterial infection and are active against Gram positive and Gram negative bacteria, including multiply resistant strains.

DC B05

IN CUNY, G D; HAUSKE, J R; HEEFNER, D L; HOEMANN, M Z; KUMARAVEL, G; MELIKIAN-BADALIAN, A; ROSSI, R F

PA (SEPR-N) SEPRACOR INC

CYC 82

PI WO 9857952 A1 19981223 (199908)* EN 137p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW
AU 9882586 A 19990104 (199921)

ADT WO 9857952 A1 WO 1998-US12706 19980618; AU 9882586 A AU 1998-82586 19980618

FDT AU 9882586 A Based on WO 9857952

PRAI US 1997-878781 19970619

AB WO 9857952 A UPAB: 20010402

Isoquinoline-indole derivatives of formula (I) are new. A, B = fused rings comprising cycloalkyl, cycloalkenyl, aryl or 4-8 membered heterocyclyl (all optionally substituted by R4 or R5); X = CR, N, NO, P or As; Y = CR2, NR, O, PR, S, AsR or Se; R, R1-R3 = H, halo, alkyl, alkenyl, alkynyl, OH, alkoxy, silyloxy, amino, NO2, thiol, alkylthio, imino, amido, phosphoryl, phosphonate, phosphine, carbonyl, carboxyl, carboxamide, anhydride, silyl, thioalkyl, alkylsulphonyl, arylsulphonyl, **selenoalkyl**, ketone, aldehyde, ester, heteroalkyl, nitrile, guanidine, amidine, acetal, ketal, amine oxide, aryl, heteroaryl, azide, aziridine, carbamate, epoxide, hydroxamic acid, imide, oxime, sulphonamide, thioamide, thiocarbamate, urea, thiourea, or (CH2)mR80; R4, R5 = a group R, but not H; m = 0-8 and R80 = aryl, cycloalkyl, cycloalkenyl, heterocyclyl or a polycycle.

USE - (I) are antimicrobial agents active against Gram positive and Gram negative bacteria, including multiply resistant strains e.g. to methicillin, ciprafloxin and vancomycin. (I) are active against Staphylococci, Streptococci, Micrococci, Peptococci, Peptostreptococci, Enterococci, Bacilli, Clostridii, Lactobacilli, Listeriae, Erysipelothrices, Propionibacteria, Eubacteria, Corynebacteria, Mycobacteria, Mycoplasma, Rickettsia and Helicobacter pylori. (I) are used for treating bacterial infections and other disorders associated with pathogenic bacteria including respiratory and pharyngeal infections, otitis, pharyngitis, pneumonia, **peritonitis**, pyelonephritis, cystitis, endocarditis, systemic infections, bronchitis, arthritis, local inflammations, skin, wound, and blood infections, conjunctivitis, and infections of surgically created vascular access e.g., in kidney dialysis. (I) are also used for treating food poisoning causing nausea, vomiting, diarrhoea and septicaemia, gastroenteritis, cystitis, tuberculosis of both humans and cattle from mycobacteria, sexually transmitted diseases e.g. gonorrhoea and trichomonas infection and typhoid fever, bacillary dysentery, and plague. (I) can be used for sterilisation of surfaces, including counter tops, tissue and cell culture media, surgical instruments, bandages, skin and mucosal surfaces including the cornea, for dermal cuts, abrasions, burns and sites of bacterial or fungal infection. (I) are used in animal breeding and livestock husbandry to promote or accelerate growth and improve feed utilisation in both healthy and sick animals including horses, cattle, pigs, sheep, and poultry and pets.

ADVANTAGE - (I) have selective toxicity to target microorganisms, with minimal toxicity to mammalian cells.

Dwg.0/0

L5 ANSWER 6 OF 17 WPIDS (C) 2002 THOMSON DERWENT
AN 1999-095304 [08] WPIDS
CR 1999-095313 [08]; 2000-431259 [36]; 2001-181513 [09]
DNC C1999-028050
TI New 2-(Indol-3-yl)quinoline compounds - active against Gram positive and Gram negative bacteria, including multiply resistant strains.
DC B02 B05
IN CUNY, G D; HAUSKE, J R; HEEFNER, D L; HOEMANN, M Z; KUMARAVEL, G; MELIKIAN-BADALIAN, A; ROSSI, R F
PA (SEPR-N) SEPRACOR INC
CYC 83
PI WO 9857931 A2 19981223 (199908)* EN 145p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW
AU 9879797 A 19990104 (199921)
NO 9906269 A 20000216 (200020)
EP 991623 A2 20000412 (200023) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
CZ 9904608 A3 20000816 (200048)

US 6207679 B1 20010327 (200119)
HU 2000003364 A2 20010628 (200143)
KR 2001014030 A 20010226 (200154)
JP 2002505689 W 20020219 (200216) 189p

ADT WO 9857931 A2 WO 1998-US12762 19980618; AU 9879797 A AU 1998-79797 19980618; NO 9906269 A WO 1998-US12762 19980618, NO 1999-6269 19991217; EP 991623 A2 EP 1998-930396 19980618, WO 1998-US12762 19980618; CZ 9904608 A3 WO 1998-US12762 19980618, CZ 1999-4608 19980618; US 6207679 B1 CIP of US 1997-878781 19970619, US 1998-45051 19980319; HU 2000003364 A2 WO 1998-US12762 19980618, HU 2000-3364 19980618; KR 2001014030 A KR 1999-712059 19991220; JP 2002505689 W WO 1998-US12762 19980618, JP 1999-504835 19980618

FDT AU 9879797 A Based on WO 9857931; EP 991623 A2 Based on WO 9857931; CZ 9904608 A3 Based on WO 9857931; HU 2000003364 A2 Based on WO 9857931; JP 2002505689 W Based on WO 9857931

PRAI US 1998-45051 19980319; US 1997-878781 19970619

AB WO 9857931 A UPAB: 20020308

2-(Indol-3-yl)quinoline compounds of formula (I) and their salts, are new: A and B = cycloalkyl, cycloalkenyl, aryl, or heterocyclic rings containing 4-8 members (all optionally substituted by R4 or R5); X = CR, N, NO, P, or As; Y = CR2, NR, O, PR, S, AsR, or Se; R, R1-R3 = H, halogen, alkyl, alkenyl, alkynyl, hydroxy, alkoxy, silyloxy, amino, nitro, thiol, alkylthio, imino, amido, phosphoryl, phosphonate, phosphine, carbonyl, carboxyl, carboxamide, anhydride, silyl, thioalkyl, alkylsulphonyl, arylsulphonyl, **selenoalkyl**, ketone, aldehyde, ester, heteroalkyl, nitrile, guanidine, amidine, acetal, ketal, amine oxide, aryl, heteroaryl, azide, aziridine, carbamate, epoxide, hydroxamic acid, imide, oxime, sulphonamide, thioamide, thiocarbamate, urea, thiourea, or 'CH2)mR80; R4, R5 = R excluding H; m = 0-8; and R80 = aryl, cycloalkyl, cycloalkenyl, heterocyclyl, or a polycycle, all optionally substituted.

USE - (I) display selective toxicity to target microorganisms, with minimal toxicity to mammalian cells. (I) are active against both Gram positive and Gram negative bacteria, including multiply resistant strains to e.g. methicillin, ciprafloxin, and vancomycin. They are used in treating and preventing bacterial infections, and other disorders associated with pathogenic bacteria. These include respiratory and pharyngeal infections, otitis, pharyngitis, pneumonia, **peritonitis**, pyelonephritis, cystitis, endocarditis, systemic infections, bronchitis, arthritis, local inflammations, skin, wound, and blood infections, conjunctivitis, and infections of any surgically created vascular access, e.g., in kidney dialysis. (I) are also used to treat food poisoning causing nausea, vomiting, diarrhoea, and septicaemia, gastroenteritis, cystitis, tuberculosis of both humans and cattle from mycobacteria, sexually transmitted diseases, e.g. gonorrhoea, trichomonas infection, typhoid fever, bacillary dysentery, and plague. (I) can be used for sterilisation of surfaces, including countertops, surgical instruments, bandages, skin, and mucosal surfaces, including the cornea, for dermal cuts, abrasions, burns, and sites of bacterial or fungal infection. In addition to clinical use for humans, veterinary uses are envisaged, as for tuberculosis in cattle above, and generally prophylactically in animal breeding and livestock husbandry, as a result promoting or accelerating growth and improving feed utilisation in both healthy and sick animals. Dwg.0/0

L5 ANSWER 7 OF 17 WPIDS (C) 2002 THOMSON DERWENT

AN 1998-583381 [49] WPIDS

CR 1997-393254 [36]; 1999-166579 [14]

DNC C1998-174550

TI Composition containing magnesium gluconate - is useful for treating allergic diseases, auto-immune diseases, septic shock and infectious diseases.

DC B05 C03

IN FLEMING, T E; MANSMANN, H C

PA (FLEM-N) FLEMING & CO PHARM; (FLEM-N) FLEMING & CO
CYC 80
PI WO 9847497 A2 19981029 (199849)* EN 26p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW
AU 9871505 A 19981113 (199913)
US 5939394 A 19990817 (199939)
ADT WO 9847497 A2 WO 1998-US8164 19980423; AU 9871505 A AU 1998-71505 19980423; US 5939394 A CIP of US 1996-588564 19960118, US 1997-844909 19970423
FDT AU 9871505 A Based on WO 9847497
PRAI US 1997-844909 19970423; US 1996-588564 19960118
AB WO 9847497 A UPAB: 19990928
Composition for treating allergic diseases, autoimmune diseases, **septic shock** and infectious diseases comprises: (a) magnesium, gluconate; and (b) one or more anti-oxidants selected from vitamin E, **selenium**, glutathione, glutathione isopropyl ester or N-acetylcysteine.

USE - The amount of magnesium gluconate is sufficient to treat diseases related to inappropriate production of lipid mediators (especially PGE2, PGD2, TXB2, LTB4, LTC4, MDA, HPETE or HETE) or cytokines (especially TNF-2, IL-1, IL-5, IL-6, IL-8 or IFN-9). The composition is useful for treating asthma, allergic rhinitis, eczema, atopic dermatitis, allergic contact dermatitis, rheumatoid arthritis, systemic lupus erythematosus, Graves' disease, immune thrombocytopenic purpura, myasthenia gravis, ulcerative colitis, Crohn's disease, scleroderma, psoriasis, infectious diseases caused by viruses, bacteria, fungi, protozoa or parasites and **septic shock** caused by gram-negative organisms e.g. Escherichia coli, Aerobacter aerogenes, Proteus mirabilis, Proteus vulgaris, Pseudomonas aeruginosa, Bacteroides species and Salmonella species.

Dwg.0/4

L5 ANSWER 8 OF 17 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 1998330461 EMBASE
TI Selenium, systemic immune response syndrome, sepsis, and outcome in critically ill patients.
AU Forceville X.; Vitoux D.; Gauzit R.; Combes A.; Lahilaire P.; Chappuis P.
CS Dr. X. Forceville, Centre Hospitalier de Meaux, Reanimation Polyvalente, 6-8 rue Saint Fiacre, 77104 Meaux Cedex, France
SO Critical Care Medicine, (1998) 26/9 (1536-1544).
Refs: 55
ISSN: 0090-3493 CODEN: CCMDC7
CY United States
DT Journal; Article
FS 006 Internal Medicine
037 Drug Literature Index
LA English
SL English
AB Objectives: To confirm early, marked decrease in plasma **selenium** concentrations in patients admitted to a surgical and medical intensive care unit (ICU), and to study this decrease according to the presence or absence of systemic inflammatory response syndrome (SIRS), sepsis, or direct ischemia-reperfusion. Design: Prospective, observational study. Settings: Collaboration between the adult ICU of a 1,100-bed general hospital and a biochemical research laboratory of a university medical center. Patients: One hundred thirty-four consecutive surgical and medical ICU patients. Interventions: None. Measurements and Main Results: In the first 31 patients, plasma and urine **selenium** concentrations were

measured by electrothermal atomic absorption spectrometry on admission and once weekly during their ICU stay. These values were compared first with severity scores, criteria for SIRS, sepsis, and organ system failure taken on admission, and then with nosocomial infection, organ system failure during ICU stay, and hospital mortality. An early, low mean plasma **selenium** concentration was observed in these patients compared with **selenium** laboratory reference values. Plasma **selenium**, measured on ICU admission, inversely correlated with Acute Physiology and Chronic Health Evaluation II or Simplified Acute Physiology II scores. Patients with SIRS had lower **selenium** concentrations than those without SIRS. Mean urine **selenium** losses were normal in the first 31 patients. Plasma **selenium** concentration was low in all patients with severe sepsis and **septic shock** (range 0.20 to 0.72 .mu.mol/L) and in those patients with ischemia-reperfusion from aortic cross-clamping (range 0.34 to 0.68 .mu.mol/L). Despite recommended specific **selenium** supplementation, plasma **selenium** concentrations remained low for >2 wks in patients with SIRS. However, there was a slight increase in plasma **selenium** concentrations in surviving SIRS patients, whereas plasma **selenium** concentrations decreased in nonsurviving patients. The frequency of ventilator-associated pneumonia, organ system failure, and mortality was three times higher in patients with low plasma **selenium** concentration at the time of admission (**selenium** .ltoreq.0.70 .mu.mol/L) than for the other patients. Conclusions: In severely ill ICU patients with SIRS, we observed an early 40% decrease in plasma **selenium** concentrations, reaching values observed in deleterious nutritional **selenium** deficiency. This prolonged decrease in **selenium** concentrations could explain the three-fold increase in morbidity and mortality rates in these patients compared with other ICU patients. The efficacy of **selenium** treatment in SIRS patients with a high gravity index score or hypoperfusion needs further investigation.

L5 ANSWER 9 OF 17 MEDLINE DUPLICATE 4
AN 1998032443 MEDLINE
DN 98032443 PubMed ID: 9365739
TI [Dilated cardiomyopathy and selenium deficiency in AIDS. Apropos of a case].
Cardiomyopathie dilatee et deficit en selenium au cours du SIDA. A propos d'un cas.
AU Constans J; Sire S; Sergeant C; Simonoff M; Ragnaud J M
CS Clinique de medecine interne et des maladies vasculaires, hopital Saint-Andre, Bordeaux, France.
SO REVUE DE MEDECINE INTERNE, (1997) 18 (8) 642-5.
Journal code: 8101383. ISSN: 0248-8663.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
LA French
FS Priority Journals; AIDS
EM 199711
ED Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971121
AB Cardiac-related death of HIV-positive patients is not rare. The etiology of AIDS-associated dilated cardiomyopathies often remains unknown, even at autopsy. We report an observation associated to a severe deficit in **selenium**. The patient had been diagnosed as HIV-positive 2 years before. He presented Pneumocystis carinii pneumonia then Cryptococcus **meningitis**. Two months later he was hospitalized for pancreatitis and cachexia. He presented global heart failure that lead to death. No microorganism was found in myocardium at autopsy but plasma **selenium** was dramatically decreased (24 micrograms/L). The deficit in **selenium** has been associated to a dilated cardiomyopathy in

non-AIDS patients. HIV-positive patients have an early decrease in plasma **selenium**, this concentration is dramatically decreased in malnourished patients. **Selenium** deficit might be the cause of some of the AIDS-related dilated cardiomyopathies and **selenium** supplementation might be useful in these patients.

L5 ANSWER 10 OF 17 MEDLINE DUPLICATE 5
AN 95231437 MEDLINE
DN 95231437 PubMed ID: 7715587
TI [Selenium and antioxidant status in various diseases].
Der Selen- und Antioxidanzienstatus bei verschiedenen Krankheitsbildern.
AU Winnefeld K; Schirrmeister W; Thiele R; Sperschneider H; Klinger G
CS Institut fur Klinische Chemie und Laboratoriumsdiagnostik, Jena.
SO MEDIZINISCHE KLINIK, (1995 Jan 15) 90 Suppl 1 7-9.
Journal code: 8303501. ISSN: 0723-5003.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals
EM 199505
ED Entered STN: 19950524
Last Updated on STN: 19970203
Entered Medline: 19950515
AB All healthy mammalian organisms are characterized by an equilibrium between the occurrence of highly reactive oxygen species and their destruction by anti-oxidants. Numerous diseases go hand in hand with a disturbance of the homoeostatis. In order to avoid or minimize the destructive effect of the oxidant stress on biological structures, therapies utilizing drugs with anti-oxidant effects are increasingly being applied. Preconditions for these therapies are a characterisation and a follow-up of the anti-oxidant status in the diseased organism. In the course of the present study **selenium**, glutathione peroxidase and malondialdehyde were determined in patients with various clinical pictures (terminal renal insufficiency, **septic shock**, high-risk gravidieties, arterioscleroisis, pulmonary carcinoma, acute myocardial infarction, test patients taking the contraceptive pill). Patients with terminal renal insufficiency and those suffering from **septic shock** syndromes clearly show a **selenium** decrease in serum and whole blood as well as a drop in the GSH-Px-activity, and increased malondialdehyde concentrations in the serum. Both are a reflection of an increased lipid peroxidation. First results of a **selenium** therapy are available for patients with therminal renal insufficiency and post-traumatically induced renal failure. The interpretation of the findings in the categories "high-risk gravidity" and "women on the contraceptive pill", which show a normal GSH-Px-activity and significantly increased malondialdehyde concentrations, seems problematic. The organism counteracts an increased lipid peroxidation with a normal plasma-GSH-Px-activity, clearly a sign of a still normal anti-oxidant potential.

L5 ANSWER 11 OF 17 MEDLINE DUPLICATE 6
AN 91245087 MEDLINE
DN 91245087 PubMed ID: 1645378
TI Plasma lipid peroxides and antioxidants in human septic shock.
AU Ogilvie A C; Groeneveld A B; Straub J P; Thijs L G
CS Medical Intensive Care Unit, Free University Hospital, The Netherlands.
SO INTENSIVE CARE MEDICINE, (1991) 17 (1) 40-4.
Journal code: 7704851. ISSN: 0342-4642.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199107

ED Entered STN: 19910719
Last Updated on STN: 19970203
Entered Medline: 19910703

AB In order to assess if an oxidant/antioxidant imbalance is involved in human **septic shock** and its outcome, we measured plasma levels of the lipid peroxides malondialdehyde--as thiobarbituric acid reactive substance--conjugated dienes and fluorescent products, together with the antioxidants alpha-tocopherol, glutathione peroxidase activity and **selenium** in 12 patients with **septic shock** and compared them with values of normal controls. At first measurements, malondialdehyde (median 3.9 mumol/l; range 2-38.8) and fluorescent products (median 21.2%; range 9.4-134) were elevated (p less than 0.05), alpha-tocopherol (median 15 mumol/l; range 7-25) and **selenium** (median 0.76 micrograms/ml; range 0.49-1.09) were depressed (p less than 0.05). Conjugated dienes and glutathione peroxidase activity were in the normal range. In non-survivors (n = 5) initial levels of malondialdehyde and fluorescent products (median 11 versus 3.1 mumol/l; 74 versus 13% respectively) were higher than in survivors (p less than 0.05) and initial **selenium** levels were lower (median 0.58 versus 0.92 micrograms/l; p less than 0.05). These results are consistent with the concept that an oxidant/antioxidant imbalance--as indicated by elevated plasma lipid peroxides and depressed antioxidants--is involved in human **septic shock** and a fatal outcome.

L5 ANSWER 12 OF 17 MEDLINE DUPLICATE 7
AN 90365026 MEDLINE
DN 90365026 PubMed ID: 2168125
TI Cardiomyopathy associated with nonendemic selenium deficiency in a Caucasian adolescent.
AU Lockitch G; Taylor G P; Wong L T; Davidson A G; Dison P J; Riddell D; Massing B
CS Department of Pathology, University of British Columbia, Vancouver, Canada.
SO AMERICAN JOURNAL OF CLINICAL NUTRITION, (1990 Sep) 52 (3) 572-7. Ref: 38
Journal code: 0376027. ISSN: 0002-9165.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW OF REPORTED CASES)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199009
ED Entered STN: 19901109
Last Updated on STN: 19901109
Entered Medline: 19900928

AB We describe a girl aged 17 y who died after a cardiac arrest secondary to **septic shock**. At autopsy, the enlarged, soft, and flabby heart showed microscopic evidence of acute myocardial infarction, myocardial edema, myocardiocyte loss, replacement fibrosis in the interventricular septum, and right and left ventricular hypertrophic nucleomegaly. The pathological diagnosis was that of cardiomyopathy due to prolonged **selenium** deficiency. The patient had been on total parenteral nutrition for 17 mo, following extensive bowel resection for intractable pain, nausea, and vomiting caused by chronic idiopathic intestinal pseudoobstruction. Seven months before death, when severe biochemical **selenium** deficiency was diagnosed, supplemental **selenium** was added to the infusion, and plasma **selenium** concentrations increased. In long-standing **selenium** deficiency, sepsis may contribute the final insult to a damaged myocardium, triggering symptomatic cardiac failure and sudden death.

L5 ANSWER 13 OF 17 MEDLINE DUPLICATE 8
AN 89114296 MEDLINE

DN 89114296 PubMed ID: 3217752
TI Trace element alterations in infectious diseases.
AU Srinivas U; Braconier J H; Jeppsson B; Abdulla M; Akesson B; Ockerman P A
CS Department of Clinical Chemistry, Lund University, Sweden.
SO SCANDINAVIAN JOURNAL OF CLINICAL AND LABORATORY INVESTIGATION, (1988 Oct) 48 (6) 495-500.
Journal code: 0404375. ISSN: 0036-5513.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198903
ED Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19890302
AB Trace elements like copper, zinc, iron and **selenium** have a significant influence on the function of the immune system. We studied plasma levels of trace elements in 53 patients with acute bacterial and viral infections. In bacterial infections (septicaemia, pneumonia, erysipelas and **meningitis**) the plasma concentrations of **selenium**, iron and zinc were decreased. Plasma copper was unchanged in patients with erysipelas, but increased in other types of bacterial infections. Although the patients with viral infections showed similar shifts of the trace elements as were observed in patients with bacterial infections, the changes were not as pronounced. A plasma **selenium** value below 0.8 mumol/l was found in only 6% of the patients with viral infections in contrast to 63% of the patients with septicaemia or 57% of the patients with pneumonia. Furthermore, in viral infections 60% of the zinc values were below the mean level of 12.8 mumol/l observed in healthy controls as compared with 90% of the values in patients with sepsis or 92% of the values in patients with pneumonia. The onset of change in trace elements occurred within a few days and persisted for several weeks. These changes seem to be non-specific and are independent of the agent causing infection. The different types of infections were followed by changes in most of the plasma proteins which are known to be associated with an inflammatory reaction. The changes in plasma proteins were most pronounced in patients with sepsis and pneumonia. Patients with sepsis having a high degree of inflammation did not show a positive correlation between the severity of the disease--as judged by plasma proteins--and the alterations of trace elements.

L5 ANSWER 14 OF 17 MEDLINE DUPLICATE 9
AN 85257604 MEDLINE
DN 85257604 PubMed ID: 4018070
TI Fatal **Selenomonas** sputigena **septicemia** probably originating from lung abscess.
AU Pinon G; Grollier G; Romet-Lemonne J L; de Rautlin de la Roy Y
SO EUROPEAN JOURNAL OF CLINICAL MICROBIOLOGY, (1985 Jun) 4 (3) 343-4.
Journal code: 8219582. ISSN: 0722-2211.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198509
ED Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850904
AB A case of fatal **septicemia** with **Selenomonas** sputigena in an immunocompromised patient is reported. The patient had a lung abscess from which the **septicemia** is believed to have originated. In contrast to the only other case reported in the literature, the isolate from our patient was characterized by very slow and difficult growth.

AB Actinomycin C, azathioprine, cyclophosphamide, indomethacin, benzydamine, phenylbutazone, aminophenazone, sodium salicylate, and some new **selenophenones** (4-alkylseleno-.alpha.-alkyl-.beta.-hexamethyleniminophenones) were tested in relation to leucine-14C incorporation into lymphocytes isolated from peritoneal exudate of rats with formaldehyde **peritonitis** and to depression of the viability of the lymphocytes. It was not possible by these means to discriminate cytostatics from antiphlogistics used in rheumatic diseases. The procedure may be a simple screening test for substances which influence protein synthesis.

AN 1997:233324 CAPLUS
DN 126:263497
TI A comparison of effectiveness of the oral and parenteral form of **selenium supplementation** in Se-deficient dairy cows
AU Zarski, Tadeusz Piotr; Debski, Bogdan
CS Department of Animal Hygiene, Warsaw Agricultural University, Pol.
SO Annals of Warsaw Agricultural University, Animal Science (1996), 32, 71-78
CODEN: AAASEQ; ISSN: 0208-5739
PB Warsaw Agricultural University Press
DT Journal
LA English
AB A comparison of effectiveness of the oral and parenteral form of **selenium supplementation** in Se-deficient dairy cows.
The investigation was performed in the region where selenium deficiency in cows had already been diagnosed. The investigation included 30 cows divided into 3 groups of 10 animals each. Cows in group I were administered a single injection of 5.0 **mg Se** in the form of Evetsel prepn. Group II received a mixt. Mineral in the amt. of 20 g per cow once a week which equalled 7.0 **mg Se** in one daily dose. Cows in group III comprised the control without any **selenium supplementation**. The administration of selenium prepns. resulted in the increase of that trace element level in the serum and milk to its proper values. However, the result of a single Evetsel administration was short-lasting. Those additived also affected pos. the health state of cows and calves and the reprodn. of cows.

AN 1996:258014 CAPLUS
DN 124:341606
TI Hematological and biochemical changes in the blood of ewes and lambs after selenium and vitamin E injection
AU Saez, T.; Ramos, J. J.; Marca, M. C.; Sanz, M. C.; Fernandez, A.; Verde, M. T.
CS Dpto. Patologia Animal (Patologia General), Facultad de Veterinaria, Zaragoza, 50013, Spain
SO Journal of Applied Animal Research (1996), 9(1), 51-60
CODEN: JANREH; ISSN: 0971-2119
PB Garuda Scientific Publications
DT Journal
LA English
AB The effect of barium selenate injection to sheep and the administration of selenium and/or vitamin E to lambs during the first days of life on the hematol. and biochem. parameters was studied. The administration of barium selenate to ewes at 50 mg/animal 3 wks before the introduction of males increased the activity of glutathione peroxidase (GSH-Px) during gestation and post-partum. Lambs born from injected ewes had a higher GSH-Px activity at the 1st week, as well as 3 wks later. Lambs injected with 3 **mg** of **selenium** and 100 **mg** of vitamin
E during the 1st week of life had high GSH-Px activity 3 wk later. The lambs injected with 100 mg of vitamin E during the 1st week of life had GSH-Px activity similar to that of the control group. Lactate dehydrogenase, creatine kinase, aspartate aminotransferase, and hematol. parameters were almost similar in all animal groups. The results show that barium selenate injection to ewes during the breeding season protects the lambs against Se deficiency in the early period of life, when they are at greatest risk of muscular dystrophy.

AN 1997:206359 CAPLUS
DN 126:224559
TI The effect of **selenium supplementation** during the early post-mating period on embryonic survival in sheep
AU van Niekerk, F. E.; Cloete, S. W. P.; Heine, E. W. P.; van der Merwe, G. D.; Wellington, A.; du Plessis, S. S.; Bekker, D.
CS Department Human Animal Physiology, University Stellenbosch, Stellenbosch, 7600, S. Afr.
SO Journal of the South African Veterinary Association (1996), 67(4), 209-213
CODEN: JAVTAP; ISSN: 0038-2809
PB South African Veterinary Association
DT Journal
LA Afrikaans
AB The effect of **selenium** (Se) **supplementation** of ewes with blood Se concns. ranging between 100-200 ng/mL on embryonic survival during the early post-mating period (days 15-35) was studied in 4 trials. In the 1st 2 trials 137 ewes were used in 1991 and 118 in 1992. After being mated as a single flock, these ewes were stratified randomly into 3 groups. One group served as a control, the 2nd was injected with 1 mL Deposel (contg. 50 **mg Se** as Ba selenate) and the 3rd group injected with 1 mL contg. 1 **mg Se** as Na selenite. During 1991, supplementation was administered immediately after the mating period. It was postponed by 14 days in 1992. Parenteral Se supplementation reduced ($p < 0.10$) the no. of ewes that lambed by > 19 % during 1991 but not during 1992. The no. of ewes producing twins was unaffected. In Trials 3 and 4 there was a consistent indication that parenteral Se supplementation of pregnant ewes between 15-35 days after mating resulted in a reduced (22-40 %) embryonic survival rate, although significant (p .ltoreq. 0.10) differences were only obsd. after the pooling of treatments receiving parenteral Se supplementation. Drenching of ewes with 50 **mg Se** as Ne selenite resulted in a similar tendency. Biochem. appraisal of the blood, kidney and liver Se status of ewes failed to reveal toxic levels. The possible mechanisms involved in impaired embryonic survival are unclear. Supplementation of ewes during the 1st month of pregnancy with parenteral Se prepns. is not recommended.

AN 1986:514027 CAPLUS
DN 105:114027
TI Effects of marginal **selenium** deficiency and winter protein **supplementation** on growth, reproduction and **selenium** status of beef cattle
AU Spears, J. W.; Harvey, R. W.; Segerson, E. C.
CS North Carolina State Univ., Raleigh, NC, 27695-7621, USA
SO Journal of Animal Science (Savoy, IL, United States) (1986), 63(2), 586-94
CODEN: JANSAG; ISSN: 0021-8812
DT Journal
LA English
AB Seventy-two Hereford .times. Simmental cows, averaging 498 kg in body wt. and 5.2 yr of age, were used in a 2-yr study to ascertain if Se-vitamin E (E) [1406-18-4] injections and winter protein supplementation would affect growth, reprodn., and health of beef cattle maintained year-round on feeds marginally deficient in Se (0.03-0.05 mg/kg). Cows received either no injection or a mixt. of 30 **mg Se** and 408 IU E injected s.c. beginning 3-4 mo prepartum and at 60-day intervals throughout the 2-yr period. Calves born to Se-E treated cows were injected with 5.5 **mg Se** and 75 IU E/100 kg body wt. at 60-day intervals beginning at 1 mo of age. Calves were born between Dec. 30 and Feb. 20 and cows were bred between Mar. 20 and May 20. Cattle grazed pasture that consisted of orchardgrass, bluegrass, and white clover during the fall, spring, and summer. During winter (Dec. 15 to May 2), cattle were fed corn silage supplemented with either soybean meal or a urea-corn mixt. Cows and calves receiving Se-E had higher whole blood glutathione peroxidase (I) [9013-66-5] activity and plasma Se concns. than controls. Se-E injections reduced calf death losses from 15.3 to 4.2% and slightly increased adjusted calf weaning wts. Hb concns. were higher in Se-E-injected supplemented calves at 1 mo of age but not at 5 or 7 mo of age. Winter protein supplementation increased calf gains during the winter, and calf weaning wts. and decreased cow wt. losses during the winter. Neither Se-E injections nor winter protein supplementation affected conception rates of cows. These results suggest that Se-E injections can decrease mortality and increase whole blood I in cattle receiving feeds that are marginally deficient in Se.

AN 1982:508848 CAPLUS
DN 97:108848
TI Production responses in **selenium supplemented** sheep in northern New South Wales. 2. Liveweight gain, wool production and reproductive performance in young Merino ewes given **selenium** and copper **supplements**
AU Wilkins, J. F.; Kilgour, R. J.; Gleeson, A. C.; Cox, R. J.; Geddes, S. J.; Simpson, I. H.
CS ARC, Tamworth, 2340, Australia
SO Australian Journal of Experimental Agriculture and Animal Husbandry (1982), 114-115, 24-8
CODEN: AAAHAN; ISSN: 0045-060X
DT Journal
LA English
AB Young Merino ewes on 5 com. properties in northern New South Wales were supplemented with Se and their prodn. was compared with untreated flock mates from weaning till first lambing at .apprx.2 yr of age. Treatments of 5 **mg Se** were given orally every 6 wk for .apprx.12 mo. Cu treatments were also included to test for a possible concurrent deficiency or interaction with Se. There were significant responses to Se in livewt. in 4 of the 5 flocks and in wool prodn. in 2 of the flocks at both shearings. Reproductive performance at 1st mating was also better in 2 flocks. There were no beneficial effects of Cu treatment nor were there any interactions with Se treatment in any aspect of prodn. measured.

AN 97:125015 PROMT
TI NUTRITION RESEARCH:
SO Food Labeling News, (26 Dec 1996) pp. N/A.
ISSN: 1064-6329.
LA English
WC 116
FULL TEXT IS AVAILABLE IN THE ALL FORMAT
AB NUTRITION RESEARCH: Selenium may help protect against cancers of the lung, prostate, colon and rectum, but has no effect on skin cancer, according to a report published in this week's Journal of the American Medical Association. Arizona University researchers studied the effect of **selenium supplementation** (200 **mg**/day) on preventing new carcinomas in patients with previous skin cancers. Compared to a control group, the selenium group had a 37% reduction in cancer incidence and a 50% reduction in overall cancer mortality. Out of almost 200 new cancer cases, the selenium group had 40% to 60% fewer prostate, colorectal and lung cancers, but there was no difference in cancers involving the skin, bladder, head, neck or breast.

AN 1999:438758 CAPLUS
DN 131:58279
TI Microelement syrup and method of its preparation
IN Sviatko, Peter; Boda, Koloman
PA Slovakia
SO Slovakia, 3 pp.
CODEN: SLXXFO
DT Patent
LA Slovak
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	SK 279128	B6	19980708	SK 1994-37	19940112
PRAI	SK 1994-37		19940112		

AB The compn. and prepn. of liq. syrup suitable for preventive and therapeutic supply of essential microelement nutrients in animals and man are described. The vehiculum can be a fruit syrup or dia syrup (for diabetic patients). The syrup contains 50-350 mg Cu, 1000-3000 mg Mn, 1500-5500 mg Zn, 10-100 mg Co, 10-80 **mg Se**, and 10-70 **mg** I/L product. The resp. microelement salts are dissolved in acetic or citric acid, the soln. is homogenized, and 25 mL of this conc. is mixed and homogenized with 750 mL fruit syrup. The syrups were tested in children (13-16 yr) and adults (16-42 yr) with good clin. results.

AN 1998:584185 CAPLUS
DN 129:230032
TI Effect of **selenium** and vitamin E **supplementation** in dairy cows
AU Zanetti, Marcus Antonio; Neunhaus, Luciana E. Domingues; Schalch, Edson; Martins, Jose H.
CS Dept. Zootecnia e Engenharia de Alimentos, USP, Brazil
SO Revista Brasileira de Zootecnia (1998), 27(2), 405-408
CODEN: RBZOFS
PB Sociedade Brasileira de Zootecnia
DT Journal
LA Portuguese
AB Forty eight Holstein .times. Zebu crossbreed cows were given dietary supplements of 5 **mg Se** as Na selenite, 500 IU vitamin E as tocopherol acetate, or 5 **mg Se** plus 500 IU vitamin E. Blood samples were collected from cows at the beginning of the expt. and after calving and from calves just after the birth. The treatment effects were verified by the serum levels of Se and by the incidence of subclin. mastitis diagnosed by the CMT test. The 5 **mg Se** supplementation during the last gestation month increased the blood serum Se levels in the cows and decreased the incidence of subclin. mastitis. Calves from the supplemented cows had serum Se levels 66% higher than controls.

AN 1996:761490 CAPLUS
DN 126:170796
TI Investigations into the influence of selenium and vitamin E on red and white blood pictures, on concentrations of several minerals and micro-elements in blood serum, and on immunologic parameters in calves
AU Bednarek, D.; Kondracki, M.; Cakala, S.
CS Dep. Cattle Sheep Diseases, State Vet. Res. Inst., Pulawy, 24100, Pol.
SO DTW, Deutsche Tieraerztliche Wochenschrift (1996), 103(11), 457-459
CODEN: DDTWDG; ISSN: 0341-6593
PB Schaper
DT Journal
LA German
AB The influence of the Se and vitamin E injections on blood and immunol. parameters of calves was investigated. After 2 injections (each 9.75 and 75 **mg Se** and .alpha.-tocopherolacetate, resp.) the calves had increased blood leukocyte counts and phagocytosis index and more NBT-pos. granulocytes. Serum contents of carotenes, vitamin A, and .gamma.-globulines increased.

AN 1996:475764 CAPLUS
DN 125:166398
TI Alpha-tocopherol, selenium and polyunsaturated fatty acid concentrations in the serum and feed of spring-calving dairy heifers
AU Wichtel, J.J.; Freeman, D.A.; Craigie, A.L.; Varela-Alvarez, H.; Williamson, N.B.
CS Department of Veterinary Clinical Sciences, Massey University, Palmerston North, N. Z.
SO New Zealand Veterinary Journal (1996), 44(1), 15-21
CODEN: NEZTAF; ISSN: 0048-0169
PB New Zealand Veterinary Association
DT Journal
LA English
AB The objectives of this study were to provide baseline data for .alpha.-tocopherol, selenium and polyunsatd. fatty acid concns. in the serum and feed of New Zealand dairy cattle, and to assess the likelihood that abnormal peroxide metab. has a role in the impaired lactational and reproductive performance noted in selenium-deficient cattle. Twenty-four Friesian heifers were randomly allocated one of four winter diets consisting of hay with or without **selenium supplementation**, or pasture and silage with or without **selenium supplementation**. A winter diet consisting exclusively of hay (.alpha.-tocopherol concn. 19 mg/kg of dry matter) resulted in a pre-calving serum .alpha.-tocopherol concn. of 1.2 mg/l compared to 4.5 mg/l for pastured heifers ($p<0.01$). The pre-calving .alpha.-tocopherol concn. for the heifers fed hay fell into the range considered deficient (<2.0 mg/l), whereas heifers fed pasture and silage remained in the range considered adequate throughout the study period. Serum fatty acid concn., and the proportion of fatty acids that were polyunsatd., were lowest in the hay-fed heifers before calving (1.0 mg/mL, 37.1% resp.), and remained unchanged following re-introduction to pasture after calving in late July and August. Serum fatty acid concn. did not increase following the re-introduction of the heifers to pasture because of the unexpectedly low fatty acid concn. (4.8 g/kg of dry matter) of the mature winter pasture. In Oct., however, the proportion of fatty acids in serum that were polyunsatd. increased (50%), as did serum .alpha.-tocopherol concns. (greater than 13 mg/l). Mean serum selenium concns. in the unsupplemented heifers ranged from 139 to 204 nmol/l, being lowest in Oct. ($p<0.01$). **Supplementation** with intraruminal **selenium** pellets (two pellets delivering about 3 **mg** of **selenium**/day) increased serum selenium concn. and glutathione peroxidase activity ($p<0.01$) whereas the type of winter diet had no effect ($p>0.05$). These results suggest that dairy cattle wintered on hay can become Vitamin E-depleted, whereas the feeding of pasture and silage should provide adequate Vitamin E. The pasture offered following calving during July and August provided a low dietary polyunsatd. fatty acid challenge, suggesting that abnormal peroxide metab. is unlikely to be an important mechanism in the impaired performance of selenium-deficient adult cattle which calve at this time of year.

AN 95:29698 PROMT
TI SELENIUM STAY REFERENCES TO BE REVOKED AS "APPROPRIATE ACTION": CVM
SO Food Chemical News, (24 Oct 1994) pp. N/A.
ISSN: 0015-6337.
LA English
WC 276
FULL TEXT IS AVAILABLE IN THE ALL FORMAT
AB The Food and Drug Administration will take "appropriate action to revoke all references" to the selenium food additive regulation stay resulting from the Sept. 13 Federal Register announcement of the stay, the Center for Veterinary Medicine said in a "selenium update," released Oct. 18.

CVM added that the agency's actions "will make it clear that the levels of selenium permitted to be added to feed are those set out in the 1987 amendments to the **selenium** food additive regulation." The maximum **supplementation** level in complete feed for chickens, swine, turkeys, sheep, cattle and ducks is 0.3 ppm. The levels for feed supplements for limit feeding and in salt-mineral mixtures for free-choice feeding for sheep and beef cattle return to those provided for by the 1987 amendments, CVM noted, adding that the osmotic selenium bolus, approved for use in beef and dairy cattle in 1989, also can be used as a source of selenium. The bolus provides 3 **mg** of **selenium** per day, the center said.

On Sept. 30, FDA appropriations legislation signed by President Bill Clinton included an amendment that suspended the stay on selenium regs until Dec. 31, 1995. The Federal Crop Insurance Reform Act, signed on Oct. 13, stated that FDA "shall not implement or enforce the stay unless the commissioner of the FDA finds that **selenium supplementation** at 0.3 ppm in complete diets is not essential to maintain animal health, is not safe to animals consuming the additive or humans consuming edible portions of **selenium-supplemented** animals ..." (See FOOD CHEMICAL NEWS, Oct. 10, Page 45).

The legislative actions removed the requirement that premix manufacturers analyze each batch of selenium premix, CVM noted.

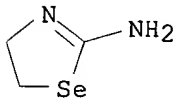
ASZ

p-9, lines 13-f

p.22 — dose disclosure
Lines 11-19

RN 15267-04-6 REGISTRY
CN 2-Selenazolamine, 4,5-dihydro- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 2-Selenazoline, 2-amino- (8CI)
OTHER NAMES:
CN 2-Aminoselenazoline
CN 2-Aminoselenoazoline
MF C3 H6 N2 Se → MW = 36 + 6 + 28 + 78.96 = 148.96
CI COM
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAPLUS, CASREACT, NIOSHTIC, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

53% se.



9 REFERENCES IN FILE CA (1937 TO DATE)
9 REFERENCES IN FILE CAPLUS (1937 TO DATE)

Examples 12, pp. 35-36 uses ASZ, etc.

p. 22 lines 11-19

For Adult Humans 5 mg — 17.5 g/day

Preferably 5 mg — 10 g/day

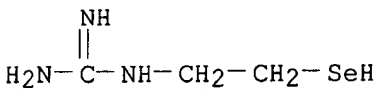
More preferably 100 mg — 3 g/day

53 mg Atomic se/day 1590 mg Atomic se/day

For a 70 kg Adult → 0.76 mg/kg/day to 22.7 mg/kg/day
This is about

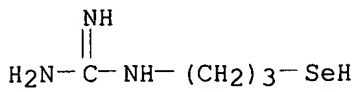
p. 9, lines 13 +

RN **57897-99-1** REGISTRY
CN Guanidine, (2-selenylethyl)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2-Selenoethylguanidine <-- SEG
MF C3 H9 N3 Se
CI COM
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAPLUS, NIOSHTIC, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)



SPG (p.9, lines 13-T)

RN **174754-71-3** REGISTRY
CN Guanidine, (3-selenylpropyl)- (9CI) (CA INDEX NAME)
MF C4 H11 N3 Se
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL



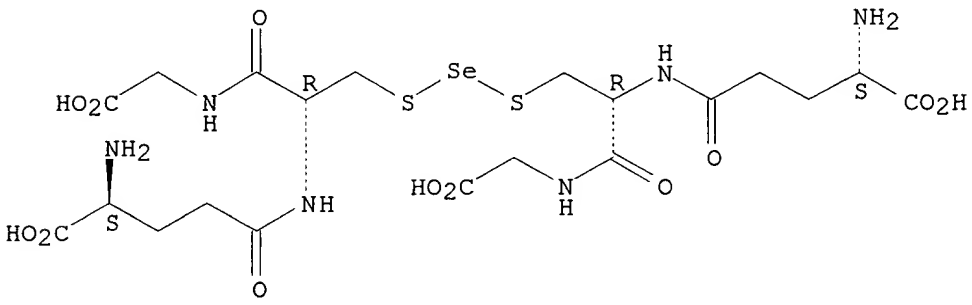
3 REFERENCES IN FILE CA (1937 TO DATE)
3 REFERENCES IN FILE CAPLUS (1937 TO DATE)

AN 2000022192 MEDLINE
DN 20022192 PubMed ID: 10554541
TI [Selenium administration in children with SIRS]. Selensubstitution bei Kindern mit SIRS.
AU Borner J; Zimmermann T; Albrecht S; Roesner D
CS Klinik und Poliklinik fur Kinderchirurgie, Universitatsklinikum Carl Gustav Carus, TU Dresden.. Jens.Boerner@mailbox.tu-dresden.de
SO MEDIZINISCHE KLINIK, (1999 Oct 15) 94 Suppl 3 93-6.
Journal code: 8303501. ISSN: 0723-5003.
CY GERMANY: Germany, Federal Republic of
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals
EM 200001
ED Entered STN: 20000114
Last Updated on STN: 20000114
Entered Medline: 20000106
AB PATIENTS AND METHOD: At the Clinic for Paediatric Surgery of the University of Dresden, in a time period ranging from 5/1994 to 12/1996, all patients aged between 1 and 16 years with severe inflammatory surgical diseases or extended scalded skin, were given an adjuvant **selenium** substitution. As control group, all patients with the same diagnosis and age treated during the months 1/1997 to 12/1998, did not receive this adjuvant **selenium** substitution. All these patients fulfilled the criteria of **"Systemic Inflammatory Response** Syndrome" (SIRS). The **selenium**-therapy group consisted of 34 patients and the control group without substitution consisted of 31 patients. The following laboratory parameters were measured on the 1st, 2nd, 3rd, 6th and last treatment day: white blood cell count, interleukin 6, C-reactive protein, fibrinogen, malondialdehyde, activity of glutathione peroxidase in plasma and level of **selenium** in plasma and whole blood. RESULTS: The initially high interleukin 6 rates declined significantly in both groups from the 2nd day on. The acute phase proteins, i.e. the C-reactive protein and fibrinogen, normalized in both groups after the 3rd day of treatment. The initial low rates of **selenium** in plasma and blood gained more rapidly a normal level in the therapy group than in the control group. On the 1st day of therapy the glutathione peroxidase activity in plasma was in both groups at the inferior limit of norm range and remained at this level in the control group for the whole observation period. In the **selenium** -substitution group on the contrary, these initial low values raised to the double as an expression of an elevated cell membrane protection. The initial significant elevated malondialdehyde rates in both groups, expressing a raised lipidperoxidation, fell down to a normal level in the **selenium**-substitution group, whereas they remained at their initial high level in the control group during the whole observation period. CONCLUSION: The substitution of **selenium** in children with S

Date no good

L2 ANSWER 1 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **33944-90-0** REGISTRY
CN Glycine, 2,2'-selenobis[L-.gamma.-glutamyl-L-cysteinyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Glutamine, N,N'-[(selenodithio)bis[1-[(carboxymethyl)carbamoyl]ethylene]]di-, L- (8CI)
CN L-Glutamine, N,N'-[selenobis[thio[1-[[(carboxymethyl)amino]carbonyl]-2,1-ethanediyl]]]bis-
OTHER NAMES:
CN Bis(glutathione) selenide
CN Glutathione, S,S'-selenobis-
CN Selenodiglutathione
FS STEREOSEARCH
DR 35178-31-5
MF C20 H32 N6 O12 S2 Se
LC STN Files: AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CHEMCATS, DDFU, DRUGU, EMBASE, MEDLINE, NIOSHTIC, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

Absolute stereochemistry.

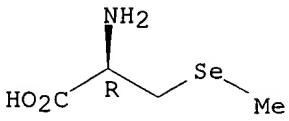


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

57 REFERENCES IN FILE CA (1967 TO DATE)
57 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L2 ANSWER 2 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **26046-90-2** REGISTRY
CN L-Alanine, 3-(methylseleno)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Alanine, 3-(methylselenyl)-, L- (8CI)
OTHER NAMES:
CN 3-(Methylseleno)-L-alanine
CN Methylseleno-L-cysteine
CN Methylselenocysteine
CN Se-Methylselenocysteine
FS STEREOSEARCH
MF C4 H9 N O2 Se
LC STN Files: AGRICOLA, BEILSTEIN*, BIOSIS, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM, DDFU, DRUGU, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

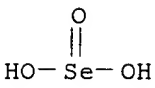
Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

64 REFERENCES IN FILE CA (1967 TO DATE)
64 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L2 ANSWER 3 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **10102-18-8** REGISTRY
CN Selenious acid, disodium salt (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Selenious acid (H2SeO3), disodium salt (8CI)
OTHER NAMES:
CN Disodium selenite
CN Disodium selenium trioxide
CN Neoselen
CN Sodium selenate (Na2SeO3)
CN Sodium selenite
CN Sodium selenium oxide (Na2SeO3)
DR 50647-14-8, 29528-97-0
MF H2 O3 Se . 2 Na
CI COM
LC STN Files: ADISNEWS, AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DRUGU, EMBASE, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PIRA, PROMT, RTECS*, TOXCENTER, ULIDAT, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (7783-00-8)



2 Na

2218 REFERENCES IN FILE CA (1967 TO DATE)
11 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
2219 REFERENCES IN FILE CAPLUS (1967 TO DATE)
24 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L2 ANSWER 4 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **7782-49-2** REGISTRY

CN Selenium (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN C.I. 77805
DR 12640-29-8, 12640-30-1, 12641-96-2, 12733-65-2, 11125-23-8, 11133-88-3, 95788-45-7, 50954-17-1, 51882-60-1, 37256-19-2, 37258-85-8, 37276-15-6, 37368-02-8
MF Se
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PHARMASEARCH, PIRA, PROMT, RTECS*, TOXCENTER, TULSA, ULIDAT, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Se

48368 REFERENCES IN FILE CA (1967 TO DATE)
1822 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
48414 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L2 ANSWER 5 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **7440-66-6** REGISTRY
CN Zinc (7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN AN 325
CN Asarco L 15
CN Blue powder
CN Ecka 4
CN F 1000
CN F 1000 (metal)
CN F 1500T
CN F 2000
CN F 2000 (metal)
CN LS 2
CN LS 2 (element)
CN LS 4
CN LS 5
CN LS 5 (metal)
CN NC-Zinc
CN Rheinzink
CN UF
CN UF (metal)
CN VM 4P16
CN Zinc Dust 3
DR 12793-53-2, 195161-85-4, 199281-21-5, 298688-49-0
MF Zn
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*,

MSDS-OHS, NAPRALERT, NIOSHTIC, PDLCOM*, PHARMASEARCH, PIRA, PROMT, RTECS*, TOXCENTER, TULSA, ULIDAT, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Zn

209481 REFERENCES IN FILE CA (1967 TO DATE)
11154 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
209637 REFERENCES IN FILE CAPLUS (1967 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L2 ANSWER 6 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **7440-57-5** REGISTRY
CN Gold (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN A 4631
CN A 4953
CN AY 5022
CN Britecote
CN Burnish Gold
CN C.I. 77480
CN C.I. Pigment Metal 3
CN Colloidal gold
CN Furuuchi 8560
CN G 1402
CN Gold 197
CN Gold black
CN Gold element
CN Gold Flake
CN Gold Leaf
CN Gold Powder
CN Palegold 5550
CN Perfect Gold
CN PH 870
CN SG 10NK
CN Shell Gold
DR 33019-35-1
MF Au
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PIRA, PROMT, RTECS*, TOXCENTER, ULIDAT, USPAT2, USPATFULL, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Au

107899 REFERENCES IN FILE CA (1967 TO DATE)
3297 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

108083 REFERENCES IN FILE CAPLUS (1967 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L2 ANSWER 7 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **7440-50-8** REGISTRY
CN Copper (7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 100RXH
CN 1100T
CN 115A
CN 1721 Gold
CN 200RL
CN 22BB400
CN 2L3GT
CN 3EC
CN 3EC-HTE
CN 3EC-III
CN 3EC-VLP
CN 3EC3
CN 3L Fire
CN Allbri Natural Copper
CN Arwood copper
CN BHN 02T
CN BHY 02B-T
CN BHY 13T
CN BHY 22B-T
CN BPF 18
CN BSH
CN BSH (metal)
CN C 100
CN C 100 (metal)
CN C.I. 77400
CN C.I. Pigment Metal 2
CN CDX
CN CDX (metal)
CN CE 1100
CN CE 1110
CN CE 115
CN CE 15
CN CE 25
CN CE 7
CN CE 7 (metal)
CN CE 8A
CN CF 78
CN CF-T 8
CN Copper element
CN Copper fulleride (CuC20)
CN Copper Powder
CN CS-F 150E
CN CT 315E
CN CU 112
CN Cu-At-W-250
CN CU-FN 10
CN Cu-HWQ
CN CuEP
CN CuEPP
CN CuLox 6010
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY

DR 133353-46-5, 133353-47-6, 65555-90-0, 72514-83-1, 195161-80-9
MF Cu
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PIRA, PROMT, RTECS*, TOXCENTER, TULSA, ULIDAT, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Cu

361041 REFERENCES IN FILE CA (1967 TO DATE)
20422 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
361386 REFERENCES IN FILE CAPLUS (1967 TO DATE)
2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

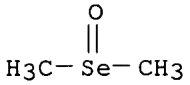
L2 ANSWER 8 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **7439-89-6** REGISTRY
CN Iron (7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 300A
CN 3ZhP
CN A 227
CN Ancor B
CN Ancor EN 80/150
CN Armco iron
CN Atomel 300M200
CN Atomel 500M
CN Atomet 28
CN Atomet 95
CN Atomiron 44MR
CN Atomiron 5M
CN Atomiron AFP 25
CN Atomiron AFP 5
CN ATW 230
CN ATW 432
CN BASF-EW
CN Carbonyl iron
CN Copy Powder CS 105-175
CN DH
CN Diseases (animal), iron overload
CN Diseases, iron overload
CN DSP 1000
CN DSP 128B
CN DSP 135
CN DSP 135C
CN DSP 138
CN EF 1000
CN EF 250
CN EFV
CN EFV 200/300
CN EFV 250

CN EFV 250/400
CN EO 5A
CN F 60
CN F 60 (metal)
CN Ferrovac E
CN FT 3
CN FT 3 (element)
CN GS 6
CN HF 2
CN HF 2 (element)
CN HL (iron)
CN Hoeganaes ATW 230
CN Hoeganaes EH
CN HQ
CN HQ (metal)
CN HS (iron)
CN HS 4849
CN Iron element
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
DR 8011-79-8, 8053-60-9, 129048-51-7, 73135-38-3, 70884-35-4, 39344-71-3, 195161-83-2, 199281-22-6
MF Fe
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PHARMASEARCH, PIRA, PROMT, RTECS*, TOXCENTER, TULSA, ULIDAT, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Fe

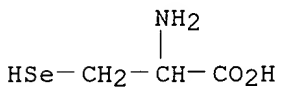
288006 REFERENCES IN FILE CA (1967 TO DATE)
17485 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
288243 REFERENCES IN FILE CAPLUS (1967 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L2 ANSWER 9 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **4371-90-8** REGISTRY
CN Methane, seleninylbis- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Methyl selenoxide (7CI, 8CI)
OTHER NAMES:
CN Dimethyl selenoxide
CN Dimethylselenium oxide
CN Selenoxide, dimethyl-
MF C2 H6 O Se
CI COM
LC STN Files: AGRICOLA, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, EMBASE, MEDLINE, NIOSHTIC, SPECINFO, TOXCENTER
(*File contains numerically searchable property data)



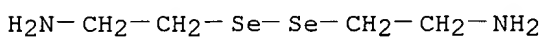
46 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
46 REFERENCES IN FILE CAPLUS (1967 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L2 ANSWER 10 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **3614-08-2** REGISTRY
CN Alanine, 3-selenyl- (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 3-Selenyl-DL-alanine
CN DL-Selenocysteine
CN Selenocysteine
DR 18312-66-8
MF C3 H7 N O2 Se
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAOLD, CAPLUS, CEN, CIN, EMBASE, HSDB*, NIOSHTIC, PROMT, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)



422 REFERENCES IN FILE CA (1967 TO DATE)
18 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
424 REFERENCES IN FILE CAPLUS (1967 TO DATE)
4 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L2 ANSWER 11 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **2697-61-2** REGISTRY
CN Ethanamine, 2,2'-diselenobis- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Ethylamine, 2,2'-diselenobis- (6CI, 8CI)
OTHER NAMES:
CN Bis(2-aminoethyl) diselenide
CN Selenocystamine
MF C4 H12 N2 Se2
CI COM
LC STN Files: ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, DDFU, DRUGU, EMBASE, GMELIN*, IFICDB, IFIPAT, IFIUDB, MEDLINE, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

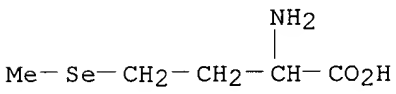


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

90 REFERENCES IN FILE CA (1967 TO DATE)

4 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
90 REFERENCES IN FILE CAPLUS (1967 TO DATE)
2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L2 ANSWER 12 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **1464-42-2** REGISTRY
CN Butanoic acid, 2-amino-4-(methylseleno)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Butyric acid, 2-amino-4-(methylselenyl)- (6CI, 8CI)
OTHER NAMES:
CN (.+-.)-Selenomethionine
CN 2-Amino-4-(methylseleno)butyric acid
CN 2-Amino-4-(methylselenyl)butyric acid
CN DL-Selenomethionine
CN dl-Selenomethionine
CN Selenium methionine
CN Seleno-DL-methionine
CN Selenomethionine
DR 2578-28-1
MF C5 H11 N O2 Se
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DIOGENES, DRUGU, EMBASE, HSDB*, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PHAR, PROMT, RTECS*, SPECINFO, TOXCENTER, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

588 REFERENCES IN FILE CA (1967 TO DATE)
7 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
588 REFERENCES IN FILE CAPLUS (1967 TO DATE)
28 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

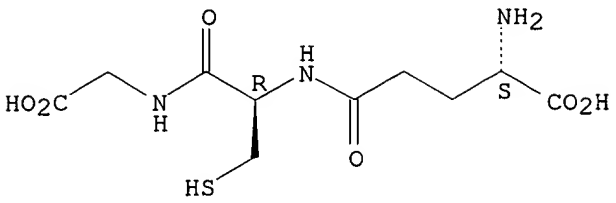
L2 ANSWER 13 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **1406-18-4** REGISTRY
CN Vitamin E (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Aquasol E
CN Covitol F 1300
CN E-Mix 40
CN Erevit forte
CN Evion
CN Fujimix E 20N
CN Hydrovit E forte
CN Irganox E 201
CN Irganox E 217
CN Irganox E 218
CN Juvela E

CN Juvela Food 500
CN MDE 6000
CN Palmvitee
CN Rocavit E
CN Rontex 201
DR 11105-14-9
MF Unspecified
CI COM, MAN
LC STN Files: ADISNEWS, AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, DIOGENES, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, NAPRALERT, NIOSHTIC, PHARMASEARCH, PIRA, PROMT, TOXCENTER, USPAT2, USPATFULL, VTB
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
13573 REFERENCES IN FILE CA (1967 TO DATE)
215 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
13607 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L2 ANSWER 14 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **70-18-8** REGISTRY
CN Glycine, L-.gamma.-glutamyl-L-cysteinyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Glutathione (8CI)
CN Glycine, N-(N-L-.gamma.-glutamyl-L-cysteinyl)-
OTHER NAMES:
CN .gamma.-Glutamylcysteinylglycine
CN .gamma.-L-Glutamyl-L-cysteinylglycine
CN 13: PN: CN1314415 PAGE: 8 claimed sequence
CN Agifutol S
CN Copren
CN Deltathione
CN Glutathion
CN Glutathione-SH
CN Glutide
CN Glutinal
CN GSH
CN Isethion
CN L-Glutathione
CN N-(N-L-.gamma.-Glutamyl-L-cysteinyl)glycine
CN Neuthion
CN Reduced glutathione
CN Tathion
CN Tathione
CN Triptide
FS STEREOSEARCH
MF C10 H17 N3 O6 S
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DRUGU, EMBASE, GMELIN*, HODOC*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PIRA, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



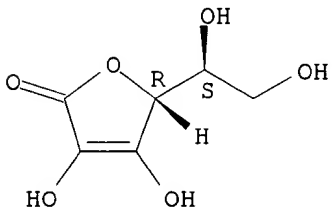
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

27903 REFERENCES IN FILE CA (1967 TO DATE)
1299 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
27947 REFERENCES IN FILE CAPLUS (1967 TO DATE)
7 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L2 ANSWER 15 OF 15 REGISTRY COPYRIGHT 2002 ACS
RN **50-81-7** REGISTRY
CN L-Ascorbic acid (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN (+)-Ascorbic acid
CN 3-keto-L-Gulofuranolactone
CN 3-Oxo-L-gulofuranolactone
CN Adenex
CN Allercorb
CN Antiscorbic vitamin
CN Antiscorbutic vitamin
CN Ascoltin
CN Ascorbajen
CN Ascorbic acid
CN Ascorbutina
CN Ascorin
CN Ascorteal
CN Ascorvit
CN C-Quin
CN C-Vimin
CN Cantan
CN Cantaxin
CN Catavin C
CN Ce-Mi-Lin
CN Ce-Vi-Sol
CN Cebicure
CN Cebion
CN Cebione
CN Cecon
CN Cegiolan
CN Ceglion
CN Celaskon
CN Celin
CN Cemagyl
CN Cenetone
CN Cereon
CN Cergona
CN Cescorbat
CN Cetamid
CN Cetemican

CN Cevalin
CN Cevatine
CN Cevex
CN Cevimin
CN Cevital
CN Cevitamic acid
CN Cevitamin
CN Cevitan
CN Cevitex
CN Chewcee
CN Ciamin
CN Cipca
CN Citrovit
CN Colascor
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
FS STEREOSEARCH
DR 56533-05-2, 57304-74-2, 57606-40-3, 56172-55-5, 129940-97-2, 14536-17-5, 50976-75-5, 154170-90-8, 89924-69-6, 30208-61-8, 259133-78-3
MF C6 H8 O6
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PDLCOM*, PHAR, PHARMASEARCH, PIRA, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

45052 REFERENCES IN FILE CA (1967 TO DATE)
1149 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
45133 REFERENCES IN FILE CAPLUS (1967 TO DATE)
12 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

US 097638700EP1

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Team: OIPEBackFileIndexing
Dossier: 09763870

Legal Date: 09-24-2003

No.	Doccode	Number of pages
1	CTNF	7
2	892	1
3	NPL	3
4	NPL	1

Total number of pages: 12

Remarks:

Order of re-scan issued on .....